ARCHIVES of OPHTHALMOLOGY

VOLUME 7 (old series Volume 64) FEBRUARY, 1932 NUMBER 2

THOMAS GATAKER

AN EIGHTEENTH CENTURY ENGLISH SURGEON WITH OPHTHALMOLOGIC LEANINGS *

R. R. JAMES, F.R.C.S.

LONDON, ENGLAND

The family of Gataker goes far back in English history. Thomas was son and heir to the Rev. Edward Gataker, M.A., rector of Mursley, Bucks, who was descended from the ancient Shropshire family of Gatacre of Gatacre. Lest any reader should wonder at the change in spelling of the family name, I may state that on the monument to Charles Gataker, great-grandfather of the surgeon, who died Nov. 20, 1680, and is buried at Hogston, Bucks, occurs the following statement: *ex antiqua Gatakerorum de Gatak-Hall in agro Salopiensi familia oriundus.*[1]

The pedigree appended, drawn up by Mr. J. Harvey Bloom, a recognized authority in such matters, shows the immediate descent, but it may be of interest to indicate a few facts about the family of earlier date than 1577.

Gatacre itself was a vill of Claverley in South Shropshire near the town of Bridgnorth. Claverley was one of a compact estate of four manors, the others being Worfield, Nordley and Alveley, which in Saxon times belonged to Algar, Earl of Mercia. The Conqueror granted them en bloc to Roger de Montgomery, his near relative, the first Norman Earl of Shrewsbury. When Robert de Belesme, younger son of Earl Roger, raised a rebellion against King Henry the First, he was promptly suppressed by that capable monarch; his castle of Bridgnorth was taken by storm and his honors forfeited. Claverley was then early in the twelfth century an escheat of Earl Robert, and as such was granted to the honor of Montgomery, the Lord of which was Baldwin de Bollers. Under the Lords of Montgomery a family of knightly rank grew up which took its name from the place of Gatacre.

The first direct mention of the Gatacre family in the public records would appear to be the entry on the Pipe Roll of 6, Henry II, at Michaelmas, 1159, where the sheriff of Shropshire is entered as owing 21 shillings of the money (de pecuniâ) of William of Gatacre. This

* Submitted for publication, Aug. 10, 1931.

1. Lipscomb: History of Buckinghamshire, London, J. & W. Robins, 1847, vol. 3, p. 384.

expression is more usually associated with some forfeiture levied on the goods of a person of poor estate, and not of knightly degree, but the sheriff paid the debt in the following year, so there must have been some cash in hand.

The name of Sir William de Gatacre occurs in connection with an action at law about half a hide of land in Shropshire a little later, and in 1194 he was one of four visors in another legal case. It was usual to appoint knights only to such an office. His son, Sir Robert de Gatacre, occurs on the Rot. Curiae Regis in April, 1200.[2]

Few English families can show an unbroken descent back to the twelfth century. The old home of the family must have been a unique specimen of architecture if one can trust what Camden said about it.

> The walls were very particular on account of their being built of a dark grey free-stone, coated with a thin greenish vitrified substance about the thickness of a crown-piece, a most effectual preservative against all bad weather, without the least appearance of any joint or cement to unite the several parts, so that it seemed but one entire piece. The hall was nearly an exact square, and most remarkably constructed. At each corner, in the middle of each side, and in the centre was an immense oak tree, hewed nearly square, and without branches, set with their heads on large stones, laid about a foot deep in the ground, and with their roots uppermost, which roots, with a few rafters, formed a complete arched roof: the floor was of oak boards three inches thick, not sawed, but plainly chipped. The whole is now entirely pulled down, and a new house built at a little distance.

Of Gataker's early life, nothing is known. I find that he was apprenticed at Barber-Surgeons' Hall on Dec. 2, 1731. The entry is as follows: "Thomas Gataker son of Edward Gataker, late of Mursley in the County of Berks (*sic*) deceased puts himself apprentice to Isaac Rider Surgeon for seven years." On Oct. 5, 1742, "Thomas Gataker late the apprentice of Isaac Rider was admitted to the Freedom by servitude."

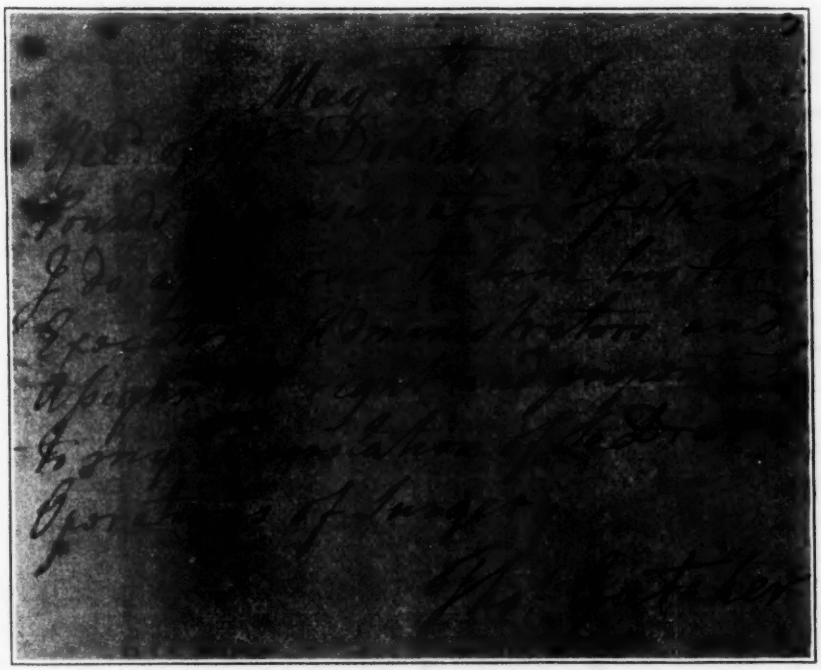
Before leaving the records of Barber-Surgeons' Hall, I may state that in Young's "Annals of the Barber-Surgeons' Company" Gataker is entered as free of the Company and Serjeant Surgeon to George III. I feel sure that this is an error; no warrant of appointment as such can be found at the Public Record Office, and the serjeant surgeons at this date were John Ranby and Sir Caesar Hawkins, both of whom outlived Gataker. I am not aware that there were ever more than two serjeant surgeons at any one time, and the office was held for life.

On June 9, 1743, Gataker was elected a governor of St. George's Hospital, with which institution he was afterward to be connected. In 1746, he married Anne Hill, daughter of Thomas Hill, of Court of Hill, Salop, and Martha Hammond. The Hills were an ancient

2. Eyton: Antiquities of Shropshire, London, J. Russell Smith, 1854, vol. 3, p. 86.

Salopian family long resident in South Shropshire, the pedigree in the Shropshire Visitation of 1623 [3] going back to the fourteenth century.

At this time he was resident in Park Place, St. James', and in 1754 he was appointed surgeon to the Westminster Hospital. He served at this hospital until 1760. It was in the year 1754 that Gataker had a controversy with Richard Guy about Plunkett's caustic.[4] I suppose it took the form of the pamphlet squabble common at this period, but no copy of any tract bearing on the subject appears in either the College of Surgeons' library or in that of the Royal Society of Medicine, while



Specimen of Gataker's handwriting.

the index catalogue of the British Museum library also proved negative. Guy was a surgeon in London who, in 1759, brought out a small pamphlet of 96 pages entitled "An Essay on Scirrhous Tumours and

3. Harleian Society, 1889, vol. 28.

4. Wadd (Mems., Maxims, and Memoirs, London, Callow & Wilson, 1827) gives the ingredients of this caustic as follows:

"Crowsfoot, which grows in low ground, one handful,
Dog fennel, three sprigs,
Crude brimstone, three middling thimbles-full,
White arsenic, the same quantity.

All incorporated in a mortar, then made into small balls, the size of nutmegs, and dried in the sun."

Cancers." A list of cases in which the author obtained cure by means of Mr. Plunkett's medicine is appended. In his introduction, Guy refers to the "necessary connection between Mr. Plunkett and myself having existed for some years which obliged me to visit him often. I knew that he pretended to a secret for the cure of cancers, but then looked upon it as a mere pretence." Finding that Plunkett effected cures, Guy bought the secret. Gataker is referred to on page 40 of this pamphlet and is there called "ingenious." Guy found Gataker's treatise on solanum to be "impartial."

On Jan. 30, 1760, William Hewitt sent in his resignation as surgeon to St. George's Hospital, and Gataker was elected to the vacancy, without opposition, at the annual general court held on Feb. 15, 1760.

In 1761, Gataker, together with Pennell Hawkins, was appointed surgeon to the household of the future Queen, i. e., the Princess of Wales. The warrant of appointment is not recorded in the books of the Lord Chamberlain, at the Public Record Office.

The minutes of the weekly board of St. George's Hospital contain very few references to Gataker. For the most part they deal with the annual payment of his subscription of 5 guineas as a governor. He seems to have been rather behind with these payments. He was a regular attendant at the board meetings up to June 3, 1768. His illness is not recorded on the minutes, and the last entry relative to him is dated March 21, 1770, when Mr. Walker paid 5 guineas as a subscription from the late Thomas Gataker, esq., for the year ending Christmas, 1768. At the election of a successor to the vacancy on the surgical staff caused by his death, the celebrated John Hunter competed and defeated David Bayford.

Gataker died in Pall Mall on Nov. 17, 1768. His will was proved in London on November 28, by his widow. I am greatly indebted to my old friend, Dr. George Peachey, the historian of St. George's Hospital, for the abstract which follows:

> Abstract of will. (P. C. C., Secker, 412). Thomas Gataker, Surgeon of Pall Mall, St. James's, Westminster. "All estate real and personal to sole use and benefit of my wife, Mrs. Ann Gataker, in full confidence that she will dispose of it to my son, Thomas and my daughter Ann Gataker, in such manner as shall be most to their advantage." His wife to be sole executrix. Dated, 29, September 1768. (signed) Tho. Gataker. Witnesses: Christopher Smith, Wm. Walker, John Haley.

While on the subject of Gataker wills, it may be of interest to add that the will of Ann Gataker, of St. George's, Hanover Square, widow of Mr. William Gataker, was proved March 26, 1772 (P.C.C., Taverner, 92). She desired to be buried with her husband in the church of Rickmansworth. She left to her neice, Hannah Willet, her house in Monmouth Street, with the remainder to Mary, wife of Mr. Thomas Hearn, linen draper, of St. Giles-in-the-Fields. To Mr. Thomas

Gataker of St. Christopher's, London, son of the late Thomas Gataker, surgeon, she left 100 pounds, at the request of her late husband, his godfather, and also the great silver salver with the Gataker arms on it and the silver tea vase.

The will of her husband was proved on Dec. 11, 1769 (P.C.C., Bogg, 415). It stated that he was of St. George's, Hanover Square, apothecary. He left all to his wife, Ann, who was to administer the goods of his late son, William. This will is dated May 26, 1753, and makes no mention of Thomas Gataker.

It occasionally happens that odd items of interest to members of the medical profession turn up in secondhand bookseller's catalogues. Such occurred in connection with the document reproduced to illustrate this article. It is an agreement between Gataker and Dodsley, the publisher, whereby all profits arising from the sale of Gataker's translation of the operations of surgery by the French surgeon, Le Dran, are sold to Dodsley. I secured it for a few shillings from a bookseller in the Midlands, and think that it must be the sole remaining specimen of Gataker's handwriting.

GATAKER'S PROFESSIONAL WRITINGS

A little octavo brochure of 86 pages was published in 1761. The title page runs as follows:

An/Account/of the/Structure of the Eye/with occasional/Remarks on some Disorders/of that Organ,/delivered in/Lectures/at the/Theatre of Surgeons-Hall./ by Thomas Gataker,/Surgeon-Extra-ordinary to Her Royal Highness the Princess of Wales/and Surgeon to St. George's Hospital,/London./Printed for R. & J. Dodsley, in Pall-Mall; and G. Hawkins, at Temple Bar, 1761./

It is dedicated to the master, governors and court of assistants of the Incorporated Society of Surgeons of London, and is composed of three lectures and an introduction, which latter displays a good deal of common sense. The lectures are for the most part purely anatomic, and the anatomy is, as is to be expected, of the gross macroscopic variety. The descriptions of the eyebrows, lids and lacrimal apparatus are elementary but sound as far as they go. The second lecture deals with the tissues of the globe. Gataker was evidently skeptical of the old view that the three coats of the eye were expansions of the optic nerve and its sheaths, "the sclera being derived from the dura mater, the choroid, from the pia mater and the retina, from the medullary substance of the nerve"; but thought that it might be "proper to retain the old opinion till a more certain one is established." His view of accommodation was that the inelastic sclera is forcibly drawn back into the orbit and pressed against the fat; whereby the humors being protruded forward, the cornea, being elastic, is rendered more or less convex.

WILLIAM GATACRE of Gatacre = ELEANOR dau. of WILLIAM MYTTON of Shrewsbury
Died 1577

THOMAS GATAKER (4th son); at Oxford 11 yrs.; Cambridge 4 yrs.; student of the M. Temple, 1553; parents sent him to Louvain and on his conversion disowned him; priest, London, 1568; chaplain to the Earl of Leicester; rector of St. Edmund, Lombard St., 1592-1593; Christ Church, Newgate, 1578-1579; d. 1592-1593 as Rector of Newington, Surrey. = MARGARET PIGOTT
Will Jan. 8, 1592-1593, pr. P. C. C. April 2, 1593 (29 Newell).

THOMAS GATAKER b. Sept. 4, 1574; B. D. 1604; Fellow of Sidney Coll., Cambridge; preacher at Lincoln's Inn, 1601; rector of Rotherhithe, 1611-1654; member of the Westminster Assembly, scholar and author; d. July 24, 1654. Will P. C. C., chaplain to Sir William Cooke, July 14, pr. July 31, 1654 (456 Alchin). M. 1st MRS. CUPPER by whom he had a son Thomas; his wife was bur. at Rotherhithe Aug. 26, 1613. M. second time MARGARET, dau. of the Rev. Charles Pinner; bur. at Rotherhithe = 3. DOROTHY, dau. of SIR GEORGE FARWELL of Hilbishop; Som: d. Aug. 15, 1627; bur. at Rotherhithe = 4. ELIZABETH

GEORGE — HENRY

ANNE — ELIZABETH

THOMAS GATAKER, to whom he left property in Rotherhithe, St. Thomas the Apostle and St. Martin Vintry

ANN d. Nov. 26, 1656 = CHARLES, son of Thomas and his 2nd wife; b. Aug. 26, 1613; at St. Paul's School; M.A. at Pembroke Coll., Oxford, 1636; incorporated at Cambridge, 1657; chaplain to Viscount Falkland; rector of Hogeston, Bucks; bur. there; d. Nov. 20, 1680; author = SARAH WHARTON of St. Martin's; Ongar.; widow Nov. 28, 1660

ELIZABETH, b. at Rotherhithe Jan. 15, 1623-1624; m. there June 2, 1640 to WILLIAM DRAPER

JOHN GATAKER, bp. Sept. 10, 1622; bur. March 27, 1627

ESTHER, bp. Jan. 10, 1624-1625; d. Jan. 20, 1626-1627

MARTHA = WILLIAM BARNARD

ANNE

THOMAS GATAKER; Clare Hall, Cambridge, M.A., 1674; incorporated at Oxford, 1676; rector of Hoggeston, Bucks; bur. in chancel = BARBARA dau. of SIR THOMAS HEBBLETHWAITE of Norton, Yorks; d. July 15, 1735 aet. 78. Will as of Swanbourne, Bucks; will dated March 16, 1734; pr. Aug. 1, 1735 (P. C. C. 168 Ducie)

THE REV. EDWARD GATAKER = M. A.; rector of Mursley, Bucks; b. Jan. 24, 1684; d. Sept. 16, 1729

WILLIAM GATAKER =

a dau. = BENJAMIN REYNOLDS; ↑ exec. to Barbara in minority of Thomas

GEORGE GATAKER = M. by license 1709 to ELIZABETH WATSON

ANNE — CHARLOTTE

EDWARD = CHARLES

THOMAS GATAKER, son and heir, surgeon to George III (P. C. C. 412 Secker) = ANN HILL dau. of Thomas Hill of Court of Hill, Salop; d. July 22, 1797

JAMES; at Westminster School 1727; legacy from Barbara

WILLIAM GATAKER of Monmouth St.; St. Giles-in-the-Fields; apothecary; will May 26, 1752; pr. Dec. 11, 1769 (P. C. C. 415 Bogg) = ANN of St. George's Hanover Sq.; bur. with husband at Rickmansworth; will pr. March 26, 1772 (92 Taverner)

THOMAS GATAKER ELIZABETH

THOMAS GATAKER of St. Christopher le Stocks and Mildenhall, Suffolk, b. 1749; d. March 16, 1844 = By license Nov. 9, 1745 to MARY SWALE

WILLIAM, d. before his father made his will 1752

THOMAS, bp. at St. Christopher Nov. 21, 1778

HENRY, bp. Oct. 10, 1780; bur. on Oct. 28, 1780

CHARLES, b. Aug. 23, 1779; bur. Sept. 19, 1780

[J. Harvey Bloom]

In dealing with the ciliary processes, he said:

It is asserted likewise that between the duplicatures of the ciliary processes fleshy fibres may be seen lying in small grooves of the membrana vitrea; but modern enquiries do not confirm the existence of these fibres.

With respect to the uses of the ciliary processes, he said:

There is also reason to believe that by their numerous villi they contribute very considerably to the secretion of the aqueous humour. One opinion of their use has been more generally received, namely, that by their contraction, the crystalline and vitreous humours are brought forward, making the cornea more prominent, as when we would view small and near objects distinctly; but as the existence of muscular fibres in the ciliary processes is by no means proved, it may be reasonably doubted whether the actions of these processes can have any power in the motion of these humours.

The circular and radial fibers of the iris are recognized, and Gataker is inclined to attribute the movements of the iris to muscular motion rather than to nervous tension and relaxation. The insensitiveness of the nerve head is alluded to, but Mariotte's name is not mentioned. The aqueous humor is secreted from the blood, and being formed constantly there must be some arrangement whereby the excess is removed. How this is done is not clearly established, but, "it is probable that part of it is taken up by the absorbent veins and part of it may transpire through the cornea."

Gataker considered the medical treatment for cataract to be perfectly useless; of the two operative methods of treatment, he adhered rather to couching than to extraction, as tedious inflammation was apt to follow the latter operation.

A good description of the extra-ocular muscles terminates the lectures, from which the following passage may be extracted:

The two oblique muscles together are by some named circumagentes and amatorii from the power which they were supposed to have of winding or rolling about the eye in the manner of looking that is commonly termed ogling.

Gataker's literary output was not confined to the eye; he was author of the following works:

Essays/on/Medical Subjects,/originally printed separately;/to which is now prefixed/An Introduction/relating to the use of/Hemlock and Corrosive Sublimate/and to/the application of Caustic Medicines/in Cancerous Disorders,/by Thomas Gataker,/Surgeon-Extraordinary to His Majesty and Her Royal/Highness the Princess Dowager of Wales, and Surgeon/to Her Majesty's Household and St. George's Hospital/London./Printed for R. & J. Dodsley, in Pall Mall./MDCCLXIV.

Overpage is a subtitle as follows:

Solanum. Use of Hemlock. Caustic Medicines in Cancerous Diseases. Use of Corrosive Sublimate. Communication of supposed discoveries or improvements and of particular or secret methods of practice. Operations for the Cataract, Venereal complaints.

The brochure on "Venereal Complaints and Their Cure" takes the form of two letters addressed to a surgeon. The first letter is dated March 1, 1754, the second, Dec. 30, 1754, and each from Park Place, St. James'. A second edition appeared in 1755.

The essay on operations for the cataract was not published. Gataker said in his introduction:

> At present, however, such a design is not to be executed with the propriety which it may be hereafter: to determine with a degree of precision necessary for public satisfaction, not only whether the operation by depression or by extraction is the better method for general practice, but whether each of them in particular instances may not have a preference, must depend in some measure on further experiments, and of course will be more properly the object of future consideration.

As Daviel's paper on extraction of cataract only appeared in 1752, it was obviously early days in which to speak of the results. Gataker must have felt that he would be skating on thin ice if he ventured on any definite expression of opinion as to the superior merits of extraction as against couching.

The "Observations on the Internal Use of Solanum or Nightshade" was first published in octavo in London, in 1757. This work would appear to have been popular for the library of the Royal Society of Medicine has copies of the third and fifth editions, both of which are dated 1757.

His largest work was his translation of "The Operations of Surgery, by M. Le Dran." The agreement with Dodsley is dated 1748, and the book was published in octavo in the following year. It contains 473 pages and 21 plates. The title is as follows:

> The Operations in Surgery of Mons. LeDran, Senior Surgeon of the Hospital of La Charité, Consultant Surgeon to the Army, Member of the Academy of Surgery at Paris and Fellow of the Royal Society. Translated by Thomas Gataker, Surgeon. With remarks, plates of the Operations, and a sett of instruments by William Cheselden esq. Surgeon to the Royal Hospital at Chelsea, and Member of the Academy of Surgery at Paris. (*Manu strenua, stabili, nec unquam intremiscente. Celsus.*) London. Printed for C. Hitch in Pater-Noster Row & R. Dodsley in Pall-Mall. MDCCXLIX.

As Cheselden died in April, 1752, it is quite likely that this was the last work on surgery in which he collaborated.

The records of the medical school of St. George's Hospital contain nothing relative to Gataker, but I find on consulting the apprentice books of the old Company of Surgeons that the following persons were apprenticed to him at Surgeons' Hall: On Oct. 2, 1755, William Walker was apprenticed to Gataker. Walker entered St. George's Hospital in March, 1760. He was later surgeon to the hospital and one of Hunter's opponents in the squabble which led to Hunter's death. It is interesting to note that Walker is one of the witnesses to

Gataker's will. I should assume that he was his medical attendant in his last illness.

On Dec. 6, 1764, Richard Bouchier was apprenticed to Gataker. I consider it likely that the Bourchier, who entered St. George's in 1767, is the same man. I have no notes about him.

On March 8, 1768, James Wilmot was apprenticed to Gataker. He entered St. George's in May of that year and was house-surgeon to the hospital in 1774, but I have no further notes about him. He paid Gataker a fee of 400 guineas.

I wish to express my gratitude to many helpers. Mr. Harvey Bloom has kindly undertaken the greater part of the research for this paper, and the pedigree and some of the wills are his work. Dr. Peachey has afforded me much assistance and has enabled me to make the paper more complete than it would otherwise have been. The secretary of the Royal College of Surgeons of England gave me every facility for examining the apprentice books, and the members of the library staff of the Royal College of Surgeons and of the Royal Society of Medicine have helped me greatly. Finally, I am much obliged to Messrs. George Pulman and Sons for the care they took in reproducing the original document. This document has been presented to the collection of autographs at the Royal College of Surgeons.

THE AFTERMATH OF CASES OF INTRA-OCULAR FOREIGN BODY*

FREDERICK A. KIEHLE, M.D.
PORTLAND, ORE.

There is an undefined feeling of exultation, a sense of magnificent accomplishment, the moment one successfully delivers a foreign body that has buried itself in an eye. The accident has descended on the patient with tragic suddenness. In less than the "twinkling of an eye" he has been visited with this dire calamity. There have ensued hours of examination, of consultation, of roentgen exposures, of suspense. Then has come the patient's entrance to hospital and to the surgical department, the preparation of the eye, the tense moment, fraught with uncertainty, when, all in readiness, the order is given to turn the switch. The tiny fragment leaps to the magnet tip, someone exclaims "There it is!" the air of suppressed excitement relaxes, and surgeon and patient as well as attendants breathe normally again. A spirit of triumphant success pervades the air. The damaged eye has been "saved"; the tragedy of a blind eye has been averted.

Yes, "hoffentlich," as the Germans say. And certain it is that the picture is a far cry from premagnet and preroentgenographic days. Tremendous forward strides have marked the progress of the knowledge of this phase of ophthalmology. To quote Shoemaker: "A few years ago failure to remove an eye containing a foreign body was malpractice. Now the reverse is true," depending on the danger of sympathetic ophthalmia. And in this connection, he adds, "I know of no disease that has so failed to make good during the war with such unlimited opportunities as has sympathetic ophthalmia due to the retention of foreign bodies."

Nothing that I may say should be interpreted as in any way discountenancing prompt removal of foreign bodies but to be honest ophthalmologists must frankly admit, to themselves and to patients, that their triumph at the moment does not, unfortunately, always spell restored ocular function. That is to say, although the damage was done at the moment of entrance of the foreign body, it is not undone at the moment of its exit.

---

* Submitted for publication, June 6, 1931.
* From the University of Oregon, Department of Ophthalmology.
* Read at the Los Angeles Meeting of the Pacific Coast Oto-Ophthalmology Society, May, 1931.

A huge bibliography on every phase of the subject of intra-ocular foreign bodies is available. Writers have dealt in extenso with the varying effects of different substances on the tissues of the eye, on the varying specificity of infecting organisms, on methods of roentgen localization, on preferential routes of removal and technic, on medico-legal aspects of the subject, as well as on aftermanagement, danger of sympathetic ophthalmia, etc. But from it all little is to be gleaned as to how much visual function remains in these eyes years afterward. It is because, suffused with the glow of apparent success, one has deceived oneself into a belief that immediate encouraging results will last? If overconfidence in the efficacy of extraction is curbed, such figures as are offered may be looked at calmly and analyzed from the standpoint of end-results.

The glaring defect in the statistics lies in omitting to note the length of time that has elapsed between the accident and the date of recording the vision. "Vision 6/6" means nothing when unrelated to the period of time elapsed. One writer details "136 cases with visual results," without giving any dates. His comments are limited to such expressions as, "result good," "eye quiet," "excellent recovery," "no subsequent visual observation recorded," and "successful," meaning doubtless that the foreign body was successfully recovered. What of these eyes in two, five or ten years?

In 1925, one of the foremost American ophthalmologists reported 250 cases of extraction of intra-ocular foreign bodies, in 72, or nearly 30 per cent, of which the eye had to be enucleated at once. The vision was 20/20 in 45 patients, in 31, or two thirds, of whom it was estimated within four months or less of the time of the accident; 20/20 in one patient at the end of four years (this was the longest period noted); 20/30 in 21 patients, 14, or two thirds, of whom were examined within four months or less; 20/40 in 5 patients, the longest period between the accident and examination being seven months; 20/50 in 4 patients, the longest period being six months, and 20/200 in 10 patients, examination being made from five months to two years after the accident. Perception of light was retained by 36 patients, two thirds of whom were examined within four months. Three of the patients who were observed the longest were followed one, two and three years, respectively.

From the pen of another well known American authority comes an analysis of another large series of cases. Four patients were reported as having "normal vision," the longest period being two years after the accident. One of 6 had 6/9 vision at the end of two years. Seven had 6/12, the longest period being at the end of a year. Two had 6/18, one at the end of six months, and the other after a year. The

same figures appeared for those with 6/30 vision. Of 3 patients observed two and one-half years, 1 counted fingers while the others had only perception of light.

I shall not weary you further with figures, except to point out the fact that an estimate at the end of six months is not particularly significant, while finger counting or perception of light is no achievement to boast of, no matter how long awaited. It is significant that the longer the period of time elapsing, the greater seems to be the diminution of vision. The last writer remarked: "The fate of an injured eye depends more upon several factors (already noted) than upon the method (route) of removal." Incidentally, as to the objection of retinal detachment after use of the posterior route, he said: "It is not borne out by cases in which clear media permit ophthalmoscopic examination years after extraction. Retinal detachment is not uncommon after injuries from foreign bodies but I have not seen it occur at the point of scleral incision."

On the 1929 program of the American Academy of Ophthalmology, a paper was presented entitled "Removal of No. 8 Lead Shot From the Vitreous with 20/20 Vision in One Case." This vision of 20/20 minus 2 was recorded twelve days after the operation. It would be interesting to know what it is today.

What happens in these eyes? Parsons said: "The tracks through the vitreous often become filled with fibrinous tissue. As this organizes and contracts the retina is pulled up and total detachment destroys vision. Or more severe irido-cyclitis may be set up and the eye shrinks." The effect on the anterior segment and on the lens need not be enlarged on. Choroidal and retinal absorption occur wherever the tissues are traumatized. In many eyes slow infection does its work with resultant atrophic iris, hypotension and atrophy of the bulbus. As a rule, there is a gradual crumbling of the ocular structure resulting in a great variety of pathologic changes: vitreous opacities, vitreous membranes, corneal maculae, subacute to chronic uveitis, pupillary membrane, retinal detachment, glaucoma, divergent eyes, sclerosis of the cornea and delayed sepsis. Frequently the best to be hoped for is an aphakic eye. When the effect is limited to the lens, perhaps the minimum of serious results are to be seen. Duane said that he "has seen a case in which except for a traumatic cataract the eye was normal 26 years after extraction." In such eyes, the eyeball is sound to be sure, but binocular vision and useful function are gone.

One must not forget that extraction means a second trauma, a new route opened through the vitreous thereby doubling the possibility of permanent damage. This consideration should, in my opinion, cause condemnation of the so-called therapeutic test by magnet. By it is meant the application of the magnet to the eye before a roentgenogram

is taken, to determine whether, by producing pain or sensation of movement, the presence of a magnetic foreign body can be determined. It has been recommended on excellent authority, but to my mind is only justified when roentgenograms are not available. Often the patient feels no "pull" till several applications of the magnet have been made from varying angles and with varying intensities of current. Each application of the current may, however, have altered the position of the foreign body within the eye and may have established a network of tracks through the vitreous. That a change in position occurs can be found by taking roentgenograms before and after the application of the magnet. Doubtless many have been disappointed in attempts to recover a foreign body when the patient has brought his roentgenograms with him, and reported previous unsuccessful attempts at removal by a surgeon. The foreign body is not found where the first roentgenograms would indicate, and a new localization is required.

But with conditions at their best and extraction "successful," the fate of these eyes is most precarious. Degeneration ensues, changes in structure follow, and one is forced to ask oneself frankly whether, functionally, the ultimate outcome is better than if the foreign body had been allowed to remain. Bulson expressed the belief that results in a series of comparative cases would not vary much where extraction had been performed and where it had not. Concluding an article entitled "Tolerance of Foreign Bodies within the Posterior Segment of the Eye," he said: "In a large percentage of cases the patient is as well off if the foreign body is left in the posterior segment as if extracted, and reported cases indicate the patient would not have fared better had intervention been adopted, as useful vision is often possible for long periods of time." In these cases also, in which the foreign body remains within the eye, Parsons said, "There are unfortunately very few records of late history. Certainly in a large number deterioration of vision occurs and even loss of sight, and it is probable that this must be regarded as the usual sequel. This occurs if not through chemical action of the foreign body, at least through chorio-retinal disorganization, or through irido-cyclitis." Hence one can hardly agree that extraction procedures are altogether unavailing, but in the face of such statistics as can yet be assembled this deduction of Bulson's is provocative of thought.

During the war observations could be made on groups of cases at desired intervals. There must today be a huge mass of information along this line available at veteran hospitals, but their staffs are occupied with more pressing and immediate duties and cannot make the desired analysis of statistics. Peacetime organizations at all comparable in authority with the military are the various state industrial accident commissions—and, of course, most of these accidents occur in industry. At least, so long as interval payments in compensation are being made, the

commissions retain a definite authority over patients and can require the remaking of visual tests at regular periods. A very real contribution to the knowledge could in time thus be obtained by careful and continued rechecks at six month intervals. A few moments suffice to determine changes in vision or in structure of the injured eye, and one could thus have in a few years dependable figures from which important deductions could be drawn.

Meantime such data as are available force one to gloomy predictions for such eyes, with the realization that their future is always hazardous. The present rule in the settlement of these cases by insurance companies and by state accident commissions is to wait not longer than six months before determining the permanent partial disability. A much closer conjecture can be made after two years have elapsed, for at best it is but a conjecture. From the standpoint of fairness to the injured man, it is much better to continue to observe him over such a period.

CONCLUSION

In conclusion, then, statistics would indicate that interest seems to be lost in this class of cases directly as the square of the time by which they recede. Despite the fact that the theme has been studied from every possible angle and although there is an abundance of data as to visual results for periods of one to six months following the trauma, only a few of these studies show the state of the eyes at later dates. Author after author is discovered to give vision without reference to dates. As well might a general surgeon boast of a high percentage of his patients with cancer alive following operation. What one wants to know is how many of them are in health two or more years afterward.

Obviously then arises the suggestion that interest be prolonged in cases of this type and data provided as to late visual results. For this purpose the patient should be informed of the importance of keeping in touch with his oculist, and he certainly would be sufficiently awake to his own interests to do so. In case of removal from the locality, he could easily be put in touch with another oculist, or in extreme cases, could be given a test chart himself and asked to report his own vision.

SUMMARY

1. There has perhaps been too free indulgence in a sense of triumph at the successful delivery of the intra-ocular foreign body, as though that dramatic event signalized the end instead of the beginning of a long drawn out struggle against slow degenerative processes.

2. All important as is prompt, dexterous and efficient technic in its removal, with the minimum of added trauma, one must envisage the fact that the damage already done the ocular structures by the entrance of

the foreign body, far from being reduced, has perhaps been increased by its emergence.

3. Statistics are wofully deficient in observations over long periods of time. Such figures as are available seem to indicate that in the majority of cases ocular function tends to decrease as time elapses, and hence that in fairness to the patient, final estimate of disability should be delayed at least two years.

4. The question should be faced squarely of whether or not, by successful extraction, one has really gained for the patient anything in vision.

5. Oculists are everywhere urged to keep their patients under observation indefinitely and to record their findings on repeated reexamination in the interest of a more exact knowledge of the ultimate status of these eyes.

BIBLIOGRAPHY

Allport, Frank: Intra-Ocular Steel Invasions Occurring in Ten Years' Practice, Am. J. Ophth. **8**:472, 1925.

Brownlie, W. B.: Clinical Study of 55 Cases of Intraocular Retention of Foreign Bodies: Result Two Years Afterward, Ophthalmoscope **14**:19; 239; 307; 471; 524, 1916.

Bulson, A. E.: Tolerance to Foreign Bodies Within the Posterior Segment of the Eye, J. A. M. A. **87**:2071 (Dec. 18) 1926.

Cridland, B.: Prognosis of Foreign Bodies in Eye and Orbit, Ophthalmoscope **14**:406, 1916.

Cross, G. H.: Removal of a No. 8 Lead Shot from the Vitreous, Tr. Am. Acad. Ophth., 1929, p. 173.

Dantrelle, D.: Foreign Bodies in Vitreous, Ann. d'ocul. **161**:835, 1924.

Derby, George S.: Intra-Ocular Foreign Body: A Surgical Emergency, Am. J. Ophth. **4**:334, 1921.

Donovan, John A.: A Foreign Body in the Lens, J. A. M. A. **83**:1136 (Oct. 11) 1924.

Duhamel, A.: Small and Nonmagnetic Intra-Ocular Foreign Bodies, Ann. d'ocul. **155**:343, 1918.

Dupuy-Dutemps, L.: Fifteen Years in the Anterior Chamber Without Siderosis, Ann. d'ocul. **161**:725, 1924.

Franklin, W. S., and Cordes, F. C.: Intraocular Foreign Bodies, Am. J. Ophth. **5**:533, 1922.

Friedenwald, Jonas S.: Pathology of the Eye, New York, The Macmillan Company, 1929.

Gibb, H. P.; Juler, F. A., and Moore, R. F.: Treatment of Intra-Ocular Foreign Bodies at Base Hospital in France, Brit. J. Ophth. **2**:564, 1918.

Haab, O.: Die Ausführung der Magnetoperation, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1922.

Jackson, Edward: Foreign Bodies in the Globe, Ophth. Rec. **17**:17, 1908.

Martin, Ancil: Magnetic Foreign Bodies in the Eye, with Report of 136 Cases, Southwestern Med. **10**:151, 1926.

Mertins, Paul S.: Intraocular Foreign Body Problems, Am. J. Ophth. **10**:414, 1927.

Mills, Harlan P., and Watkins, W. W.: Localization of Foreign Bodies in or About the Eye, Southwestern Med. **11**:488, 1927.

Mills, Lloyd; and Jeancon, Etta: Unrecognized Magnetic Intra-Ocular Foreign Bodies and Their Legal Aspects, Arch. Ophth. **4**:194 (Aug.) 1930.

Morax, V.: Intra-Ocular Foreign Body of Forty-Six Years' Duration, Am. J. Ophth. **5**:533, 1922.

Morgan, James A.: Steel Embedded in the Eye Six Years Without Symptoms, Am. J. Ophth. **7**:532, 1924.

Morse, Ashley W.: Electromagnet Extraction of Foreign Bodies from the Eye, Northwest Med. **25**:667, 1926.

Parsons, J. H.: Diseases of the Eye, ed. 5, London, J. & A. Churchill, 1926.

Petit, Paul: Conséquences tardives des corps étrangers, Ann. d'ocul. **153**:273, 1816.

Römer, Paul: Textbook of Ophthalmology in the Form of Clinical Lectures, translated by Foster, New York, Rebman Company, 1912-1913, vol. 2, p. 373.

Schwartz, W. A.: Copper Foreign Bodies in the Eye, with a Report of 37 Cases, Southwestern Med. **11**:504, 1927.

Shoemaker, W. T.: Penetrating and Perforating Wounds of the Eyeball with Diagnosis and Treatment of Retained Foreign Body, Am. J. Ophth. **2**:590, 1919.

Wagenmann, A.: Fremdkörpeverletzungen, Verletzungen des Auges, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1921.

DIPLOPIA AND OTHER DISORDERS OF BINOCULAR PROJECTION

ALEXANDER DUANE, M.D.*
NEW YORK

DIPLOPIA

Disorders of binocular fixation are regularly accompanied by corresponding disorders of projection. Of these the most common is double vision or diplopia.

Pathologic diplopia, due to derangement of binocular coordination, is to be distinguished from physiologic diplopia, which is the regular accompaniment of normal vision and which is a potent factor in the production of stereopsis.

The two conditions are differentiated as follows:

Physiologic Diplopia	Pathologic Diplopia
The object of fixation appears single and is distinct.	The object of fixation appears double, one image being distinct, the other indistinct.
The only objects seen double are those that are obviously further or nearer than the object of fixation. The nearer the objects are to the later, the less double they appear, and those that are alongside it appear single.	Most of the objects in the field of view appear double, but particularly those that are close to the object of fixation and are alongside it.
The diplopia is hardly ever recognized spontaneously and rarely causes confusion.	Diplopia often obtrudes itself on the notice and often causes confusion and discomfort.
If an object is seen double, both images are indistinct, and are also shadowy or ghostlike, so that if one is in direct line with the object of fixation, the latter can be seen through it.	When an object is seen double, one image has the natural appearance of the object itself; the other is more or less indistinct and shadowy.
The diplopia is not affected by shifting the gaze laterally or vertically, provided the convergence is unaltered.	The diplopia is often increased or diminished by shifting the gaze sideways or up and down.
The diplopia can be made to disappear at once by changing the convergence, so as to fix an object nearer or more remote, as the case may be.	Frequently the diplopia remains when the convergence is altered.

Both physiologic and pathologic binocular diplopia are distinguished from monocular diplopia by the fact that in the two former one of

---

* Dr. Alexander Duane died on June 10, 1926. Mrs. Duane has courteously permitted the publication of certain chapters from the book on "Ocular Muscles," on which Dr. Duane was so industriously working and which he had nearly completed at the time of his unfortunate end. These chapters represent the views of an authority who devoted his life to scientific research in a field to which he had so ably contributed.—THE EDITORS.

the double images at once disappears when either eye is shut. When this happens, the remaining single image, if indistinct before, at once becomes distinct, provided the eye has good vision.

In pathologic diplopia the distinct image of the objects of fixation, i. e., the one seen by the fixing eye, is called the real image; the indistinct image seen by the nonfixing eye is called the false image.

When double images are very close together, they overlap so that only the outlines of the object appear doubled. In this case the patient is often not even aware that he sees double, but complains only of seeing indistinctly or says that every object has a shadow.

VARIETIES OF DIPLOPIA

Double images may be variously arranged. Thus they may be side by side (lateral diplopia), one above the other (vertical diplopia) or tilted so as to form an angle with each other (torsional diplopia).

In order to be clinically useful, any description of diplopia must not only state whether it is lateral, vertical or torsional, but also indicate which image belongs to the right eye and which to the left. Thus one may say that a patient shows diplopia with the image belonging to the right eye below and to the right of the other. To avoid a long drawn out statement like this, there are the following convenient descriptive terms:

Relation of Right-Eye to Left-Eye Image	Name of Diplopia	Abbreviation
	Lateral diplopia	
On right	Homonymous	DH
On left	Crossed (or heteronymous)	DX
	Vertical diplopia	
Below	Right	DR
Above	Left	DL
	Torsional diplopia	
Tipped to right (or left tipped to left)	Intorsional	DI
Tipped to left (or left tipped to right)	Extorsional	DE

CAUSATION OF DIPLOPIA

A nonphysiologic double vision is ordinarily caused by a deviation of one of the eyes from the fixing position. Regularly and often with great precision, the kind and amount of diplopia correspond to the kind and amount of deviation.

The explanation of the diplopia occasioned by a deviation of the eyes is like that of physiologic diplopia. In both cases the dominance of convergence sensations make each eye project as if from the midline binoculus.

To explain this let us consider the case of estropia. The essential element in this is a primary or secondary excess of convergence which makes both eyes tend to converge nearer than the point of fixation. Since rarely, if ever, is a convergence effort unequally distributed between the two eyes, both eyes in this case converge excessively and to the same extent. Hence instead of being directed at the point of fixation *C* (fig. 1), both are turned in equally to the nearer point *A*. In that case, as shown in figure 1 in a previous article,[1] *C* will appear in homonymous double images; *K* and *P*, on either side of *C*. But *A* will not appear clear since the eyes are not accommodated for it but for *C*, and the visual desire to get a clear image of *C* will compel one of the eyes to move in such a way as to get its image on the fovea. Suppose it is the left eye that does this. To do it, it will have to swing to the left until it points directly at *C*, and the image of the latter hence falls on the fovea *a*. This leftward swing is a conjugate movement shared equally by the right eye. Hence when the left eye has swung to the left through the angle *ALC*, the right has swung also to the left through an equal angle. The two eyes therefore now have the positions shown in figure 2, the left eye pointing toward *C*, the right deviating from the latter twice as far as before and pointing toward *G*.

As the left eye swings to the left, the projected image *K* of the object *C* swings to the right until it finally gets to occupy *C* and appears then clear and sharp. At the same time, the projected image *P* of the right eye swings equally to the right and occupies the position *H* (fig. 2). As it does this it becomes more indistinct, since its retinal counterpart now is twice as far removed from the fovea as before.

An esotropia thus produces an homonymous diplopia, corresponding in degree to the amount of deviation.

It will be observed that the squinting angle, which was *KOP* (fig. 1) when the eyes were both converged on *A* and which is now *CRG*, has remained unchanged in this process of rotation. It will also be observed that the double images produced, viz., *C* and *H*, in character and in size are essentially like those produced in physiologic diplopia. Thus the images *C* and *H* in figure 2 differ from the images *K* and *P* in figure 1 only in the fact that the former have both been shifted over to the right by an amount equal to half the distance between them. Moreover, like *K* and *P* in figure 1 in the previous article and figure 2, *C* and *H* in figure 2 are produced by projecting from the binocular; *C* represents the image formed on the fovea *a* in the left eye, being projected as from *f* in the bivisual line *OC*, and *H* represents the image formed on *p* in the right eye, being projected as from the corresponding point *q* of the binoculus. Thus in the case of an ocular deviation, as in the case of binocular fixation, all images formed on the retina of either eye are

1. Duane, A.: Binocular Vision and Projection, Arch. Ophth. **5**:735 (May) 1931.

projected as if both eyes were united in the binoculus and their visual axes coincided with the bivisual line. And this is so because in one case, as in the other, the two eyes are subjected to an equal converging effort. Such an equal converging effort forms, as has been seen, the postural basis of midline projection.

The only real difference between the conditions represented in figure 2 and those shown in figure 1 in the previous article is that in the former the diplopia affects the object of fixation while in the true physiologic diplopia shown in figure 1 in

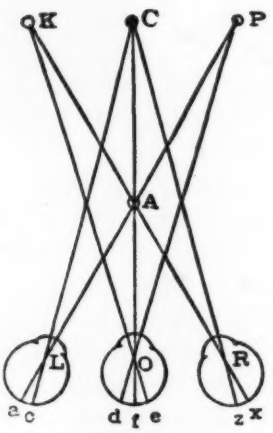


Figure 1

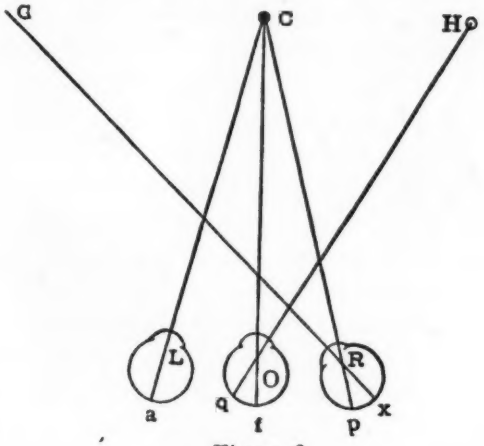


Figure 2

Fig. 1.—Both eyes are converged equally as in the case shown in figure 2, but, owing to the excess of converging power, both are directed at an object, *A*, nearer than the object of fixation, *C*. *C* is thus seen in homonymous double images, *K* and *P*. The notation is the same as in figure 1 in the previous article, to which reference should be made for an explanation of diplopia.

Fig. 2.—Same case as in figure 1, but both eyes have now swung to the left, so that the left eye is pointing at *C*. As it receives the image of *C* on the fovea, *a*, it projects this image as if from *f*, *i. e.*, in the bivisual line and out to *C*. The right eye is turned in twice as far as before, namely, to *G*, 30° to the left of *C*. It thus receives the image of *C* on the point *p* 30° to the left of the fovea, *x*. But, in spite of this deviation, the right eye continues to follow the law of binocular projection because it is still under the domination of the sensations set up by convergence, which, as shown in figure 1 of the previous article, is equal for the two eyes. It hence, like the left eye, projects as if its visual line, *RG*, were coincident with the bivisual line, *OC*. Consequently the object *C*, which forms its image on *p*, is projected as from the corresponding point, *q*, of the binoculus, i. e., out of *H*. If the right eye should happen to lose completely its association with the left and escape entirely from the domination of convergence impulses, it would project simply according to the laws of monocular vision; that is, it would not project the image falling on *C* out to *H*, 30° to the right of the bivisual line *OC*, but would project it 30° to the right of its own visual line, i. e., out to *C*. In this case there would be no diplopia, but a conflict of superposed images leading to suppression. If the right eye has not escaped wholly from the domination of convergence and is in part governed by the impressions afforded by conjugate impulses, which rule monocular vision, the image on *p* will be projected somewhere between *C* and *H;* i. e., the diplopia will still be present, but will be less than the deviation (incongruous diplopia).

the previous article it does not. In the latter case there is seen a pair of shadowy double images accompanying a third, single and distinct image, which represents the object of fixation, while in pathologic diplopia there are two images, one shadowy, the other distinct, of the object of fixation itself.

Quite analogous is the explanation of the crossed diplopia produced by a divergence of the eyes.

Diplopia due to paralysis of one of the eye muscles finds essentially the same explanation. As long as the impulses arising from equal convergences are dominant, the object fixed by either eye will be regarded as lying in the bivisual line, and an object the image of which lies at some outlying point like *p* (fig. 2) will be projected from the corresponding point *O* of the binoculus; i. e., it will appear to bear to the bivisual line *OC* the same relation that it actually bears

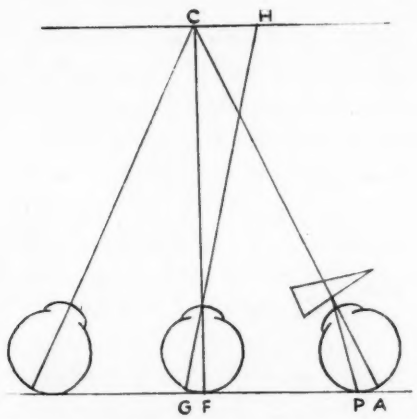


Fig. 3.—Diplopia produced by a prism.

to the monocular visual line *RG*. In other words, the effect on the projection of the equal converging impulses sent to both eyes is so strong that each visual axis *LC* and *RG*, is still conceived of as occupying the midline position, *OC*, no matter how either axis may actually be dislocated. And it makes no difference in this regard to what cause this dislocation of the axis may be due, whether excessive or faulty convergence, excessive or faulty divergence, or paretic weakness of one of the muscles. The convergence impulse in all these cases, remaining equal in the two eyes, controls the projection.

The production of a vertical diplopia is similarly explained. Suppose there is a right hypertrophia, the right eye being directed 10 degrees above the other. The right fovea then will be 10 degrees too low. Hence the image of an object, *A*, fixed by the left eye will be formed 10 degrees above the fovea in the right eye. If the latter were acting alone, this image would be projected by it 10 degrees above the point at which the eye is actually directed, i. e., to its proper place. But if the

right eye is acting with the left under the influence of convergence, the image of *A* will be projected 10 degrees below the bivisual line and below the visual plane, while the left eye image of *A* will be projected properly along the bivisual line and in the visual plane. There will thus be a right diplopia. Obviously, in the case of right hypotropia, the conditions being reversed, there would be left diplopia. So also in left hypotropia there would be a left and a right diplopia.

Take finally the case of a torsional diplopia. Suppose that the vertical meridian of the left eye is truly vertical and that of the right is tilted 10 degrees to the right. Then a vertical line will appear vertical to the left eye, but to the right eye will appear tilted to the left; that is, there will be an extorsional diplopia.

Vertical diplopia, it will be observed, does not occur as a result of physiologic action. Homonymous and crossed diplopia occur both as physiologic and as pathologic conditions. Torsional diplopia does not occur as a physiologic process when the eyes are converged.

It will be observed that in every case when diplopia is produced by the deviation of an eye the image formed in the deviating eye (false image) is projected in a direction opposite that in which the eye itself is deflected. Thus if the eye is deflected to the left, the false image is deflected to the right; if the eye is deflected up, the image is deflected down; if the vertical meridian is tilted to the left, the image is tilted to the right, etc.

The condition that causes the diplopia may not be a deviation due to a motor anomaly, but a mechanical displacement of the eye produced by pressure, with the finger or a limitation of movement produced by pterygium, etc.

DIPLOPIA PRODUCED BY PRISMS

As is known, rays of light passing through a prism are deflected toward the base of the latter. Accordingly, if when the two eyes are both sighting the same object, a prism, base in, is placed before the right eye, the light coming from the object no longer falls on the fovea, but is deflected to the left so as to impinge on a point distant, say, 10 degrees from the fovea. The right-eye image, therefore, is projected by the binoculus not from *f* as before, but from *q*, 10 degrees to the left, just as is the case when the right eye is deviated inward (fig. 2). *C* will thus appear double, the left-eye image being in its proper place at *C*, the right-eye image at *H*, to the right of *C*. A prism, base in, thus produces an homonymous diplopia.

Similarly, a prism, base out, produces a crossed diplopia; one base up before the right eye or down before the left produces a right diplopia, and one placed the opposite way produces a left diplopia.

A diplopia produced by prisms obviously falls in the same category as that produced by a pathologic deflection of the eyes. In both cases diplopia is due to the fact that an image which to produce single vision should fall on the fovea actually falls on some extrafoveal point, and the only difference is that in a deviation of the eyes the fovea is deflected away from the image; in diplopia produced by prisms, the image is deflected away from the fovea.

FORE-AND-AFT AND OTHER ANOMALOUS FORMS OF DIPLOPIA

In lateral diplopia, whether produced by deviation, or by prisms, the two images, instead of appearing side by side, may appear one in front of the other. This fore-and-aft diplopia may be pushed by special artifice (Frölich), and occasionally it occurs spontaneously, although the conditions present are such as should produce images set side by side. I saw this once in a case of divergence paralysis in which the homonymous double images of a candle seen at a distance of 30 inches (76.20 cm.) without any especial background were alleged to be at different distances, the right being nearer. When, however, the images were projected on a plane background, they appeared side by side.

In quite the same way, in conditions causing vertical diplopia, the image that should be higher may seem on a level with the other, back of it or even lower (reversed diplopia). This occurs particularly when the eyes are directed down, and is due partly to the character of the background on which the images are projected, and partly to other factors (M. Sachs and Frölich). If, for example, one sees two images, one above the other, of a lighted candle placed on the floor, instead of seeing the upper image floating in the air, one usually projects it to the floor, so that it appears to lie behind the other. If, again, one places the candle on a stair and standing at the top of the stairs looks down at it, the upper image will actually appear below, since one will then project it on one of the stairs farther down.

OVERCOMING DIPLOPIA

Diplopia produced by a deviation of either eye will be reduced and made to disappear by turning that eye in the direction opposite to that in which it is deviated, i. e., toward the false image. Thus if the right eye is turned in, as shown in figure 2 so that p, the retinal image of C, falls too far to the left of the fovea, all that is necessary to restore single vision is for the eye to swing to the right, until the fovea, x, and the image, p, coincide.

In this case the right eye does not merely swing to the right, while the left stands still, since independent motions of this sort rarely, if ever, occur. What happens is that both eyes diverge and at the same

time both are swung to the right, so that the left eye, receiving an equal impulse to move to the right and to the left, remains steady, and the right eye, receiving a double impulse to go outward, arrives at the proper position. The movement by which the diplopia is redressed is thus just the opposite of that by which it is produced.

So too, if the right eye is diverged, single vision is restored if both eyes converge and then both swing to the left. If the right eye is elevated 10 degrees above the other, it comes down 5 degrees and the left rises 5 degrees, by a movement of convergence in a vertical direction (the see-saw movement of Maddox), and at the same time both make a conjugate movement of 5 degrees downward. Thus the left or fixing eye remains stationary simply because it is under the influence of two equal opposing forces, and the right executes a double movement under the influence of two equal powers acting in the same direction.

Diplopia produced by prisms is corrected in an entirely similar way by a combination of a converging, diverging or see-saw action (vergence) with a conjugate movement (version). The net result of the combination is that the eye behind the prism moves toward the apex of the prism in order to produce single vision. Such a movement will always be evident when the eyes are actually overcoming the diplopia produced by a prism. If then I feel uncertain as to whether or not a patient is overcoming the diplopia, I look for this movement of the eye behind the prism. So too, if the patient, with the left eye screened, is looking at the light with the right eye and a prism is interposed, base down, before the latter, both eyes should be observed to turn up, as the seeing eye in order to fix the light follows the image upward. This is the basis of Graefe's test.

In overcoming diplopia the eye rotates about a center that is several millimeters back of the nodal point. Hence the angle of rotation is, theoretically speaking, slightly less than the angular amount of the diplopia itself or the angular displacement produced by a prism. But the difference is so small as to be entirely negligible.

The ability to overcome diplopia, whether resulting from a deviation of the eyes or produced by a prism, is dependent partly on the readiness with which the special movement required is made and partly on the development of the fusion faculty. In some cases, this ability is extraordinary in amount, deviations of 20 or 30 degrees of arc (in exceptional cases even more) being overcome by muscular effort. This is especially true of the crossed diplopia caused by exotropia or by prisms, base out. The ability here is dependent on the power of convergence, when, as is regularly the case, it is very strong. The ability to overcome homonymous diplopia due to esotropia or to prisms, base in, is much more limited, and the power of overcoming vertical deviations or prisms, base up and down, rarely amounts to more than 2 degrees of arc. In exceptional cases, however, it is much greater. I saw a case of paresis of the superior rectus muscle in which in the primary position there was a diplopia of 7 degrees (13-14), which the patient could and usually

did correct without any compensatory movement of the head. In another case of paresis, the patient, a girl of nervous disposition, not only could overcome a considerably vertical diplopia due to the deviation, but also at times would exaggerate the latter to a considerable amount by voluntary or involuntary effort.

Cases of this sort have a wide range of fusion and are evidence of unusual ability to perform verging and conjugate movements. Opposed to them are the cases in which the range is small, or even cases in which there is no tendency to single vision.

CORRECTION OF DIPLOPIA BY PRISMS

Diplopia may also be relieved by a prism. If the diplopia is caused by a prism, it can obviously be relieved by superposing another prism of equal strength set in the opposite direction.

If the diplopia is caused by a deviation of one eye, all that one has to do is to put over that eye a prism of the proper strength with its apex directed in the way in which the eye is deviated. Suppose, for example, the eye is deviated to the left. The false image formed by that eye is deviated to the right. One must get it back to the left. One can do so by placing a prism before the eye with its apex to the left, since objects seen through a prism always appear deflected toward the apex.

It is not actually necessary that the prism be placed before the deviating eye. The same result is effected if it is placed, but in the opposite direction, before the fixing eye. Thus a right hypertropia of 5 degrees can be corrected equally well by a prism, apex up, before the right eye or, apex down, before the left. In the latter case, the left eye moves down to fix the image deflected by the prism, and the right, moving down with it, arrives at the fixing position.

EFFECT OF TILTING THE HEAD IN MODIFYING DIPLOPIA

Diplopia may be diminished or modified not only by turning the eyes, but also by turning the head. This is done particularly in cases of paralysis, in which the head is turned in such a way that the eye, being carried out of the field of action of the paralyzed muscle, no longer deviates. It is also accomplished by tilting the head to one shoulder. As has been seen, when this is done the eye, in normal cases, projects as though its equator had remained horizontal instead of rotating with the eye, as it actually does. When diplopia is present, the effect of a projection of this sort is to make the right-hand image, whether formed by the right eye or the left, descend and the left-hand image rise, when the head is tilted to the right, and vice versa.

Diagrams may be constructed for the various deflections of the eyes—vertical, lateral and compound. The essential deductions from such diagrams are as follows:

If one admits that when the head is tipped to either shoulder projection takes place, as if the equator of the eye had remained horizontal, then:

1. If there is either a simple lateral diplopia or a combined vertical and lateral diplopia to start with, tilting the head to the right shoulder always depresses the

right-hand image, no matter whether the image belongs to the right eye or the left; tilting the head to the left shoulder has the reverse effect. In this way a vertical diplopia is developed if it does not already exist, and a vertical diplopia originally present is made to increase or decrease. The lateral diplopia, originally present, decreases whenever a vertical diplopia is developed by tilting the head, or when by the same means it is made to increase. On the other hand, it increases if by tilting the head a vertical diplopia originally present is made to decrease.

2. If there is a simple vertical diplopia to start with, tilting the head to either shoulder diminishes it and produces an associated lateral diplopia, which is crossed or homonymous, according to circumstances.[2]

This statement would mean that a patient with a combined crossed and right diplopia or with a combined homonymous and left diplopia would tilt the head to the right in order to diminish the vertical diplopia and deviation, while one with a combined homonymous and right diplopia or a combined crossed and left diplopia would tilt the head to the left. The patient would tilt the head in precisely the opposite way if he altogether ignored the information afforded by postural projection and were governed simply by visual guides.

As a matter of fact, a good many patients do thus diminish a vertical deviation and diplopia by habitually tilting the head, and in such patients the diplopia shows a considerable increase as soon as the head is set straight. In many of these cases a purely postural projection evidently obtains; i. e., the patients follow the rules given. In other cases, the head is tilted in the opposite fashion, as if visual projection predominated. This seems to happen especially in congenital paralysis. The explanation of these cases is difficult, as they do not in other regards seem to project in any way different from the usual.

WHY DIPLOPIA IS INSTINCTIVELY AVOIDED

To most persons diplopia is very disagreeable, causing confusion and sometimes vertigo, so that those in whom it occurs make constant instinctive efforts to avoid it. This is apparent even in infants. The most natural method of doing this is to place the eyes in the right position by muscular effort, thus overcoming the diplopia. Attempts of this kind, however, often cause considerable strain and, if not quite successful, enhance the confusion, because the closer the double images are brought together without actually uniting, the more troublesome they become. Especially disagreeable, I think, are the conditions in which the compensatory effort sometimes suffices and sometimes does not, so that there is now single vision, now vision with the images overlapping and again, at times, perhaps a wide and changing diplopia. Particularly annoying are the sudden variations in the diplopia of paralysis when the eyes are turned in different directions. For such a varying diplopia, the patient finds it difficult to make allowance, so that he does not ignore it as easily as he does one that is constant in amount. Moreover, it is much more likely than the latter to cause vertigo and other disagreeable symptoms.

---

2. The diplopia is crossed, if in a right hyperphoria the head is tilted to the right, or in a left hyperphoria is tilted to the left; in the reverse case, it is homonymous.

When the patient finds that he cannot overcome the diplopia readily or without undue strain, he may obviate it by closing one eye. This is done by infants affected with congenital deviations. It is also done by adults who have a recently acquired paralysis. This, of course, is but a temporary means of relief. In many such cases the attempt is made to obviate diplopia by tilting the head. If all of these means fail, the patient often helps himself by increasing the deviation so as

Diplopia, Its Causes and Correction

	Caused by		Corrected by		
Kind of Diplopia	A Deviation of	A Prism Placed with Apex	A Double Movement of Eyes (Vergence + Version)	A Prism with Apex	Head Movement
Homonymous	Either eye in	Out before either eye	Movement of divergence combined with a parallel movement of both eyes, to right or left	In before either eye	Turning of head in direction in which diplopia increases
Crossed	Either eye out	In before either eye	Movement of convergence combined with a parallel movement of both eyes, to right or left	Out before either eye	Turning of head in direction in which diplopia increases
Right	Right eye up or left eye down	Down before right eye; up before left eye	Movement of left supervergence combined with parallel movement of both eyes up or down	Up before right eye; down before left eye	Turning of head in direction in which diplopia increases, or more often tilting of head to one shoulder
Left	Left eye up or right eye down	Up before right eye; down before left eye	Movement of right supervergence combined with parallel movement of both eyes up or down	Down before right eye; up before left eye	Turning of head in direction in which diplopia increases, or more often tilting of head to one shoulder
Intorsional	Turning of either or both vertical meridians in	.....	Movement of divergence of vertical meridians (distorsion)		
Extorsional	Turning of either or both vertical meridians out	.....	Movement of convergence of vertical meridians (contorsion)		

to separate the images widely and thus render them less confusing. This seems to be a process that is effective in the development of many convergence and divergence anomalies. Similar in effect are the changes that in many cases of paralysis develop in the opponents of the paralyzed muscles, rendering the deviation and hence the diplopia more and more comitant and consequently less confusing.

Finally, a patient may learn to suppress one of the two images and thus get rid of all the disagreeable results of diplopia. In so doing, of course, he also loses stereoscopic vision.

A diplopia of long standing (particularly one that is due to a congenital deviation), especially one that is fairly constant in amount, often causes little or no discomfort. In fact, such diplopia may cause so little trouble that the patient is not aware of the double images until they are demonstrated by the eye surgeon's examination.

SUMMARY OF RELATIONS OF DIPLOPIA TO DEVIATION

The statements made in the preceding sections may be summarized as in the accompanying table.

DIPLOPIA ALWAYS DUE TO FALSE PROJECTION

It must be noted that binocular diplopia, whether physiologic or pathologic, is always the result of a false projection. In monocular vision the eye almost invariably projects correctly; the combination of visual and postural impressions enables it to do this. In binocular vision the two eyes, if acting in a normal way, project correctly the object looked at with even more accuracy than in monocular vision. They also project correctly a few outlying objects, which form images on corresponding points, or which at any rate occupy the horopter. But all other objects are projected out of their true place, and the same is true of all objects seen by a deviating eye. The false projection in both cases is due to the preponderance of the postural impressions derived from convergence over those derived from conjugate movement; i. e., it is due to the fact that when one looks with both eyes one locates objects with reference to the midline instead of with reference to the visual axes of the eyes themselves.

If postural impulses derived from conjugate movement chance to reassert themselves and partially or wholly to replace those derived from convergence, the diplopia diminishes (becomes incongruous) or disappears (suppression).

USUAL CORRESPONDENCE OF DIPLOPIA AND DEVIATION; EXCEPTIONAL CASES (INCONGRUOUS DIPLOPIA AND SUPPRESSION)

Diplopia and the deviation that causes it are usually quite correspondent. Thus, if one discovers by objective examination that a patient has an exotropia of 15 degrees, one should, if double images can be elicited at all, expect to find a crossed diplopia of the same amount, the double images being 15 degrees apart. Moreover, if the exotropia increased definitely in one direction of the direction of the gaze or if it increased as the object of fixation was approximated to the eyes, one should expect to find the diplopia increasing in the same way. Thus, like the deviation that causes it, a diplopia may be comitant or noncomitant, periodic or continuous and constant or intermittent. This is true not

only of an actual or manifest diplopia, but also of the masked or latent diplopia, which is revealed by such tests as the parallax, Maddox rod or phorometer.

Naturally, this correspondence is not always exact. The patient may give faulty statements regarding the diplopia, because he observes inaccurately or because when tested he is overcoming part of the deviation by unconscious effort. Again, the deviation itself not infrequently varies in amount from one moment to another.

However, apart from these comparatively slight and variable discrepancies one sometimes finds differences that are considerable and constant between the subjective and the objective tests. Thus, the deviation as measured by the latter may be considerably greater than the diplopia revealed by the former. In this case the diplopia is said to be incongrous; or if, as sometimes happens, it is opposite to what one would expect (e. g., crossed instead of homonymous), it is called paradoxical.

In still another series of cases, in spite of the existence of a manifest deviation there is no diplopia, and often none can be elicited even with the aid of apparatus (red glass, Maddox rod, prisms). The absence of diplopia in these cases is due to a suppression of the image formed by one eye, so that the patient, although apparently using both eyes, sees with but one eye at a time.

INCONGRUOUS DIPLOPIA

Incongruous diplopia exists when the deviation found by the objective tests (screen, etc.) differs in amount from that shown by the diplopia or other evidences afforded by the subjective tests (red glass, phorometer, Maddox rod, parallax).

Not all cases in which such differences are found are examples of incongruity. For example, an exotropia of 15 degrees may have been found by the screen test and directly afterward a crossed diplopia of only 10 degrees. This does not necessarily mean that there is an incongruity of 5 degrees; i. e., that there is that much real difference between the subjective and the objective findings. It may be that when the diplopia was tested, the patient was using extra convergence, perhaps because of extra accommodative effort, and hence had actually diminished his deviation by 5 degrees. Measurements frequently do vary in this way because of some change in the circumstances in which they are made. One must therefore regard as cases of incongruity only those in which the discrepancy between objective and subjective tests not only is marked, but is found repeatedly. Particularly is one certified of the diagnosis if the discrepant tests are made under conditions as nearly alike in all respects as possible. In this regard the parallax test is especially convincing, since it is made simultaneously with the screen

test under conditions precisely alike. If, then, these two tests differ, it is fairly good evidence that the subjective and objective measurements do disagree and that there is incongruity.

A review of forty-two cases of incongruity which I have studied reveals the following facts:

1. Incongruity may occur in all kinds of deviations, lateral or vertical, alternating or monocular and paretic or comitant. Thus, among my forty-two cases there were twenty with esotropia, four with esotropia with vertical deviation, nine with exotropia, five with exotropia with vertical deviation and four with deviation, mainly vertical.

Incongruity may even occur when there is no deviation, but this, I believe, happens only in case there has been a squint that has been relieved by operation.

2. It may affect both vertical and lateral projection, or it may affect one and not the other. In my forty-two cases, the incongruity seemed purely lateral in thirty-two, both vertical and lateral in six (but surely vertical in only three of them) and purely vertical in four.

3. Presenting rather a special type are the cases in which, owing to a large central scotoma, the patient fixes more or less uncertainly with an eccentric portion of the retina. In these cases there may be no true deviation when both eyes are open and yet apparently quite a marked one when the eyes are alternately screened. For when the good eye is covered, the amblyopic eye shifts its direction, not in order to get a deviated fovea in line with the object, but in order to shove a non-deviated fovea to one side so as to get the image on a more sensitive portion of the retina. If the patient is aware of the movement he is making, i. e., knows how the eye is pointing, he will project the object more or less in its true place, just as he would in monocular projection. There will be little or no deviation shown by the subjective tests, but a considerable one will be shown objectively. There will thus be a marked incongruity. If, however, the patient is unaware of the movement he is making, he will project as in ordinary binocular vision, and the object will appear displaced as it should be, in the direction opposite to the movement of the eye. Then there will be no incongruity, as subjective and objective tests will agree. It may happen that in the same case both forms of projection occur (on these different points, see case 1).

4. In incongruity the subjective deviation is regularly less than the objective (negative incongruity) and is of the same kind; that is, an exotropia is associated with a crossed diplopia, but of less amount, an esotropia with a smaller degree of homonymous diplopia, etc. One meets, however, with a number of exceptions to this rule. The diplopia may be greater than the objective (positive incongruity) or may be of

an opposite kind, being homonymous when there is an exotropia or crossed when there is an exotropia (paradoxical or perverse diplopia). But, so far as my experience goes, both positive incongruity and a paradoxical diplopia are not really different from a negative incongruity, but are simply stages in the transformation that a negative incongruity undergoes as the result of operation or otherwise.

Transformation of a perverse diplopia occurs not infrequently. It causes trouble after operation, the diplopia that was scarcely obvious before becoming marked and often annoying (see cases 2, 3, 5, 6 and 9). It would seem as if this were particularly the case when a diplopia is converted by operation into one of an opposite kind, e. g., when an homonymous diplopia becomes crossed. This should lead one in operating in cases of squint with incongruity to be careful not to carry the operation beyond that point at which a paradoxical diplopia develops. In other words, one seeks to reduce the diplopia and not the deviation itself to the lowest terms.

5. Some of these cases exhibit antipathy to single vision, or, at any rate, a tendency to change suddenly from a crossed to an homonymous diplopia (see cases 2, 3, 8 and 9). In such cases, one should expect the diplopia to persist after operation, no matter how closely one had approximated to the proper correction. Both cases that show this antipathy to single vision and those that do not may exhibit suppression. In other cases, one finds more or less tendency toward fusion and even well marked stereopsis (cases 4, 5, 7 and 8), and in some by cultivating fusion it seems possible to develop stereopsis and relieve the incongruity.

ILLUSTRATIVE CASES OF INCONGRUITY

CASE 1.—*Central Scotoma and Eccentric Fixation; Variable Incongruity (Vertical and Lateral).*—Juliette P., aged 21, was operated on at the age of 7 for esotropia which had been observed since she was 2 years old. The right eye was always amblyopic. Vision in the right eye was 9/200; that in the left, 15/12. Examination under homatropine showed: right, +4.00 (approximately); left, +2.75 + 0.50 cyl. The right eye showed central scotoma of 15° or more in diameter, corresponding to the area of choroidal atrophy above the macula. On alternate covering, the right eye diverged considerably or at times dropped and diverged slightly (apparent screen deviation indicates S 4°, RH 3.5°). But when a corresponding corrective prism combination (prism 8$^{\Delta}$, base out, and 7$^{\Delta}$, base down) was placed before the right eye, marked contrary parallax obtained (PX and PL). Furthermore, when the crossed parallex was abolished by prism of 20$^{\Delta}$, base in (which also made the object of fixation look in its true place), S by screen was marked. There was thus apparently a marked incongruity. This was also apparent from the fact that with a DX of 11 or 15° (with the Hirschberg test) there was only an inconsiderable exotropia. Yet later tests seemed to indicate that the subjective and objective tests were more or less in agreement.

CASE 2.—*Esotropia; Lateral Incongruity Changing in Character After Operation; Antipathy to Single Vision.*—Esther R., aged 13, had had noticeable squint since the age of 4. She had been using glasses for six years (present glass, +1.00),

The right eye showed esotropia for distance and alternating esotropia for near. She received: right, +1.50 + 2.00 cyl.; left, +1.75 + 0.50 cyl., representing correction found under homatropine (which, however, was really an undercorrection). Vision in the right eye was 15/30; that in the left, 15/15+. With this glass esotropia, alternating for distance, and alternating or left for near, S 11-16° (Sc), 4-5° (Ph and Rg); S′ 16°. Tenotomy was performed on the internal recti of both eyes, with moderate stretching. After this diplopia (DX) obtained, which was always greater than the deviation indicated by the screen, which was slight. One month after operation there was slight esotropia for distance, binocular fixation for near, with normal convergence near point. X 2-3° (Sc), 5-7° (Rg). Examination under homatropine now showed: right, +2.50 + 0.25 cyl.; left, +2.75 + 0.50 cyl. With a decidedly undercorrecting glass, the eyes showed very little deviation. Ten years later the patient reported that diplopia frequently occurred, but was not troublesome. Ortho (Sc) X 0.5° (Ph); DX with prisms of from 0° to 5Δ, base in, and 6Δ, base in, gave DH; no prism produced single vision. With the amblyoscope, fusion and stereopsis obtained. Practice with the amblyoscope excited a tendency to overcome the diplopia, although the fusion range was still small.

Case 3.—*Esotropia; Insufficiency of External Rectus; Negative Lateral Incongruity with Antipathy to Single Vision; Operation Causing Paradoxical Diplopia.*—Emma F., aged 20, had had esotropia since early childhood. She habitually saw double, especially for objects from 50 to 60 cm. from the eyes. Vision was 20/20 in both eyes. There were slight hyperopia and astigmatism (shown under homatropine); S (alternating esotropia); 14° (Sc), 12° (perimeter), 4-5° (Ph, Rg). S′ at 30 cm., about same as S, but within 30 cm. S′ diminishes. At 13 cm. the esotropia turned into an esophoria, which continued to 8 cm., when insuperable DX developed; Eo R<n. by 2 mm. and by perimeter = 36°, whereas Eo L = 45°; Ei R = 60°.

Operation was performed for advancement of the external rectus of the right eye. After this Eo R. was 43° or better; four weeks later, S 4° and S′ 5° (Sc); DX 4° (Rg) and a little more by amblyoscope. The patient did not get SVV with any prism, a prism of 7Δ, base in, giving DX and one of 8Δ, DH.

Case 4.—*Esotropia; Lateral Incongruity.*—Edna C., aged 9, had had esotropia since the age of 2. For two years she had worn a somewhat undercorrecting glass.

Vision was 16/15 in each eye, with right, +2.75 + 1.00 cyl. 90°; left, +2.62 + 0.75 cyl. 90°; Rfc, S (alternating esotropia) 7° (Sc), 3° or less (P and Ph); S′ 9° (Sc), 0° (P and amblyoscope). Fusion and stereopsis were obtained with the amblyoscope.

Case 5.—*Esotropia; Lateral Incongruity; Operation, Causing Paradoxical Diplopia.*—Arthur Y. T., aged 9, had had alternating squint since the age of 2. Vision in the eyes was 19/15, with right, + 0.25 + 1.00 cyl. 30°; left, + 0.25 cyl. 180° (examination under homatropine); S (alternating esotropia) and S′ 30° more or less (Sc), 11-17° (P and Ph); Ei, R and L<n by 2 or 3 mm. No fusion was noted with the amblyoscope.

Resection of the external rectus and tenotomy of the internal rectus of the right eye were performed, followed by practice with the amblyoscope; three months later, S (tropia) 4° (Sc.); X up to 11° (Ph); S′ (tropia or phoria) 9° (Sc); SVV alternating with DX (amblyoscope and Rg). With the amblyoscope and stereoscope, fusion and stereopsis obtained.

Case 6.—*Esotropia with Lateral Incongruity; Operation Producing Paradoxical Diplopia.*—Helen A. R., aged 50, had undergone repeated operations on the muscles

of the eyes many years ago, and about three years ago another operation, evidently an advancement of the left internal rectus, had been performed. She saw double in looking to the left. Examination under homatropine showed: right, +0.75 + 0.50 cyl.; left, + 0.50 + 0.75 cyl.; without glasses, S 2-3° (Sc), 15° (Ph.) and >15° (P) X′ 11° (Sc) and about the same in Er, but << to 4.5° in El; in moderate El, DH changing in Ef to SVV. With the amblyoscope, there was binocular vision, but no fusion; with bar reading, there was imperfect binocular vision.

It was evident that previous to the last operation the patient had had an exotropia. This is inferred not only from the character of the operation (advancement of the internal rectus), but also because of the nature of the incongruity; that is, before the operation she had an exotropia of a certain amount and a crossed diplopia probably of 10 or 12° less. By the operation, the exotropia was reduced to zero or transferred to a trifling inward deviation, but the slight crossed diplopia was changed into a large homonymous diplopia. A negative incongruity of one kind is thus changed into a positive incongruity of the opposite kind. On the other hand, for near points the patient retained her negative incongruity. Thus in the eyes—front position the outward deviation (X) was marked, but was not associated with crossed diplopia; in looking to the left, this outward deviation diminished, with the production of an homonymous diplopia. This diminution of the deviation and the consequent development of a paradoxical diplopia, in the eyes-left position, were evidently caused by the operation.

CASE 7.—*Esotropia; Hypotropia from Paresis of Right Superior Rectus; Incongruity, Lateral and Sometimes Vertical.*—Vincenzio D., aged 10, had had noticeable squint for several years. Examination showed: right, + 3.50 + 1.00 cyl.; left, + 4.00 + 2.00 cyl.; vision, 20/15 in both eyes (examination under atropine); without glasses, S and S′ 13° (Sc), less by Rg LH 5-6° (Sc); DL about same, although varying; in Eu and r, R drops (well marked lagging); with full correction, less 0.25 D, S 10° (Sc); slight or nil (M and Rg). With the amblyoscope, there were fusion and little or no deviation; LH 5° (Sc) and LH′ 9° (Sc) but 0° (M).

CASE 8.—*Esotropia; Lateral Incongruity; Antipathy to Single Vision.*—In Charles B. S., aged 18, vision in each eye was 17/15, with right, —0.50 + 1.00 cyl.; left, —0.25 + 0.75 cyl.; X 5-9° (Sc); S (P) of 15°; S or X (M). With the amblyoscope, there was either slight S or slight X, but never fusion. After practice with converging exercises and with the amblyoscope and stereoscope, five months later, the eyes showed: X 5° (Sc), O (Ph); X′ 9° (Sc), 1.5° (Ph); S′ 2.5° (amblyoscope), PeB 105 mm. There was stereopsis with the amblyoscope.

CASE 9.—*Exotropia; Lateral Incongruity; Operation Causing Paradoxical Diplopia.*—Immanuel J., aged 18, had had exotropia since the age of 2. At the age of 12, operation (probably tenotomy) had been performed on one eye, and at 16, tenotomy had been performed on both external recti. Since then diplopia and rather marked asthenopia had been present. Vision was 18/15 in each eye. Examination under homatropine showed: right, + 0.12 + 0.50 cyl.; left, + 0.12 + 0.37 cyl.; without glasses, X 3° (Sc and P), S (Ph) 3-4°; DH 2-3° but never fused images with any prisms; at another trial with X of 2° (Sc), DH of 15° or 16° (Rg). With the amblyoscope he brought the images together only when the tubes were converged 30° or 40°, and then he lost one image (suppression). On correction of the refraction, vision began to improve, and the patient began to overcome the diplopia.

CASE 10.—*Alternating Comitant Vertical Deviation with Vertical Incongruity.*—Mary M., aged 18, had had deviation (upturning) of the right eye since the age of 9 months. She had never had spontaneous diplopia. The vertical deviation alternated. She was aware of this, and could shift the fixation voluntarily from one eye to the other, so as to get at will either hypertropia of the right eye or hypotropia of the left eye. With correction of the slight myopia and astigmatism, vision in the right eye was 15/20+; that in the left, 15/15; RH 15° (Sc) (same whether correcting prism was put before right or left eye) and moderate X. Yet with 10$^{\Delta}$ prism, base down, before the right eye, she got DL 10$^{\Delta}$ (i. e., a net RH of 0°) and DH, if the right eye fixed, and DL 2$^{\Delta}$ (i. e., a net RH of 4°) and DX, if the left eye fixed. With weaker prisms there was alternating vision, and alternating vision was also obtained on the tangent curtain at 0.75 meter. With the amblyoscope binocular vision obtained, but when the images were brought close together, one of them was suppressed. There was comitant hyperphoria, i. e., no lagging of either eye anywhere, and the screen deviation was about the same in all parts of the field of fixation.

In this case there was a vertical incongruity of from 11 to 15°, with little or no evidence of lateral incongruity.

SUPPRESSION

In a large number of cases of squint, especially a continuous, comitant squint, the squinting eye does not see the object that the other eye is fixing. For this object it acts just as if it were closed. This suppression of the image in the squinting eye affects not only the object of fixation but also objects nearer and more remote, which, therefore, like the object of fixation itself, appear single. It may be said that a person affected with suppression shows neither pathologic nor physiologic diplopia. Because of this lack of physiologic double images and the lack of the other helps afforded by binocular vision, the patient has an inadequate sense of depth and distance, so that objects look flat, not solid (absence of stereopsis).

Suppression not only occurs in squint, but may be produced artificially in any eye, especially by conditions that for the time render the sight of one eye different from that of the other. Moreover, it may be produced voluntarily, e. g., by those who, when looking through a microscope or ophthalmoscope with one eye, leave the other open and yet do not see with it. Furthermore, if a permanent suppression has once been produced in a case of squint, it regularly, although not invariably, persists after the squint has been relieved, and the two eyes once more fix normally together.

One might suppose that the suppression would be most apt to occur when the two eyes differed greatly in vision. But this is true chiefly of the suppression that develops when the two eyes are performing binocular fixation. In this case the more the distinctness of one retinal image is reduced, the more readily does suppression occur. But if the two eyes are not fixing the same object, even greater inequality of

vision and great differences in the character of the retinal images will not necessarily produce suppression. In fact, in many such cases, diplopia is readily recognized.

This is seen particularly in artificial suppression. Thus if one looks at two similar pictures in an amblyoscope and dims one of them by any device, e. g., a red glass, both images will be recognized as long as they are widely separated, but when by bringing the arms of the amblyoscope into proper position one makes the images overlap, the dim one disappears. One thus gets suppression alternating with diplopia, according to the position of the tubes. The same thing is seen not infrequently in a diplopia occurring naturally. Such a diplopia may be apparent as long as the images are far apart, but when by prisms or other artifices the images are brought close together, one of them tends to disappear and does actually disappear when it overlaps its fellow (cases 9 and 10). The tendency to suppression in these cases varies a good deal. In some instances, the diplopia is still recognizable when the double images are quite close together; in other cases only when they are far apart, and then but fitfully.

The extent to which a one-sided amblyopia is compatible with the recognition of double images is often extraordinary.

Thus, in Rosamond G., with esotropia with paresis of the inferior rectus, there was no suppression, although the vision in the fixing eye was 16/15 and that in the deviating eye but 10/200 or less. In this case diplopia was readily recognized with the red glass and also with the amblyoscope. A more extreme case was that of a young man who a number of years before I saw him had received a pistol shot wound of the orbit causing choroidal ruptures and paralysis of the superior rectus. The vision in the good eye was normal, but with the injured eye he could only count fingers at a few feet. Yet he readily recognized the considerable diplopia caused by the paralysis.

Suppression like that just noted, which occurs when the eyes are both adjusted for the same object, but does not occur when one of the eyes is made to deviate, may be called orthophoric, or, since it affects the fovea, foveal suppression. It is to be distinguished from strabismic or peripheral suppression, which occurs when one eye is deviating and which is usually present under all conditions, no matter how the squint is made to vary either spontaneously or by artifice and even when it is reduced to nothing (complete suppression). For the development of strabismic suppression it is by no means necessary that the vision in the two eyes should be different; in fact, the most typical forms are found in cases in which the two eyes have the same vision and each alternately fixes while its neighbor suppresses (alternating squint). Many cases of monocular squint show a like constant suppression. Between these cases of strabismic suppression, which is constant under all conditions of deviation, and the orthophoric suppression, which is present only when the two eyes are in or near the fixing position, there are, as has just been noted, transition forms in which suppression alternates to a varying degree with diplopia.

Strabismic suppression is associated chiefly with comitant squint, especially with the continuous and constant forms. This is not due simply to the fact that squint of this type is usually of long standing. Suppression does not generally occur in noncomitant squint, no matter how long it has lasted. True suppression, for instance, is rarely seen in congenital paralyses, in which one would most expect to find it. And suppression apparently develops in a comitant squint only when it has ceased to be periodic and particularly when it has ceased to be intermittent. All this is tantamount to saying that in a condition which would naturally give rise to diplopia suppression does not usually occur so long as the position of the double images with relation to each other varies notably, either when the eyes are carried in one direction or another or when they are converged or diverged. This constitutes a striking clinical fact, the importance of which will be indicated later.

A hypertropia or hypotropia, probably because it is usually noncomitant, at least at its outset, is less likely to be associated with suppression than is an esotropia or exotropia.

Not to be confounded with suppression is the condition in which a patient fails to see double, because the image of the object at which he is looking happens in one eye to fall on a portion of the retina made insensitive by disease (retinitis pigmentosa, hemianopia, etc.).

Suppression may be demonstrated by making the patient hold a red glass before one eye and look at a light. If he has binocular single vision, he will see a single light with a reddish tinge; if he has simultaneous vision but does not fuse, he will see two lights, one deep red, the other white, and if he has suppression he will see but one light, red or white according to the fixing eye. If this test indicates suppression, the absence of stereopsis can generally be demonstrated with the stereoscope, the amblyoscope or by other means.

Continuous uniocular suppression tends to produce amblyopia in the deviating eye. This result does not occur in alternating squint, in which each eye suppresses while it deviates, but regains practice in fixation and in foveal vision while its fellow is deviating (alternating suppression). Alternating suppression is found not only in alternating squint, but also sometimes when there is no deviation. It occurs, for example, in normal eyes looking with the stereoscope or amblyoscope at two objects, equally distinct but either decidedly unlike in contour or of complementary color. In this case the observer sees first one image, then the other, but not both at once. In alternating squint, when the vision of the two eyes is quite different, alternating suppression may cause confusion owing to the sudden change in the character of the images when first one eye, then the other, is used.

Suppression effects only objects lying in that portion of the field of vision which is common to the two eyes. Hence objects that are so far in the temporal field of the squinting eye that they cannot be seen by the other are recognized by the former. Accordingly, even in suppression a man's peripheral field (recognition of dangers to one side of him) is practically normal (Fuchs). Moreover, the object that forms its image on the fovea of the deviating eye is not suppressed,

but this object is not seen double since its image formed on the retina of the fixing eye is suppressed (regional exclusion). Regional exclusion can readily be demonstrated by placing the patient's head in a perimeter rest and directing him to fix the zero mark on the perimeter arc and at the same time to observe without following a small light carried along the arc. If a red glass is placed over the fixing eye, the light will appear red until it reaches a point directly in line with the deviating eye, when it will suddenly become white.

DEFECTS OF FUSION WITHOUT SUPPRESSION

In many cases, especially in squint, the fusion faculty is not absent but simply more or less deficient. Thus the patient may be able to fuse images but does not get stereopsis, or he may fuse images of one kind and not others. Again, he may get fusion under ordinary conditions, but cannot do so if the least obstacle is interposed. In such cases, which occur especially in neurasthenia, hysteria and organic disease of the brain, the mere act of placing a red glass, or the weakest prism, before one eye, or in still other cases of covering the eye for a moment, suffices to evoke an insuperable diplopia.

In some persons this abolition of the fusing power may be carried to such a point that the patient never by any adjustment of the eyes or with any prism gets union of the double images, so that, for example, a prism of 5^{Δ}, base out, produces an homonymous, and a prism of 6^{Δ}, base out, a crossed diplopia, but no prism produces single vision. This condition, which von Graefe called antipathy to single vision, is found in squint and seems to occur particularly in conjunction with incongruity (cases 2, 3, 8 and 9).

EXPLANATION OF SUPPRESSION AND INCONGRUOUS DIPLOPIA

Suppression and also incongruous diplopia may, I think, be most plausibly explained by supposing that when these conditions obtain the deviating eye escapes more or less from the influence of the postural influence of convergence and even in binocular vision acts, in some regards at least, as though governed by the impressions set up by conjugate movement, i. e., by the impressions that dominate monocular sight.

To make this clear, one has only to take such a case as that shown in figure 2 and inquire what would happen if the deviating eye *R* projected the object *C*, as it would do if the other eye were shut and *R* remained pointing toward *G*. In that case *R* would project the object *C*, not with reference to the bivisual line, *OC*, but with reference to its own visual axis *RG*. By impressions conveyed by the muscles acting on it and effecting a conjugate movement, *R* would become aware that it was directed to the left and, in fact, actually pointing toward *G*. *G* would appear in its proper place, and *C* would be projected to the

right of *A* and through the angle *RAC*—therefore also to its proper place. If *R* projected thus when both eyes were open, there would appear two coincident images of *C*, one seen by the right eye, the other by the left. The condition, however, would not be the same as in ordinary binocular single vision, for while the left-eye image of *C* would be distinct, the right-eye image, being located on an extrafoveal part of the retina, would be indistinct. Whenever such images of the same object are superposed, the distinct image tends to extinguish the other, until it finally disappears altogether. This would be the case here no matter whether the actual deviation of *R* was greater or less. No matter how this deviation varied, *R*, by getting from monocular—i. e., conjugate—muscular sensations a notion of its true direction, would project the object fixed by the other eye to its proper place, and when thus projecting it would lose sight of it (suppression).

Now suppose that the reversion to the monocular type of vision (projection by conjugate instead of convergent impulses) was not complete, as in the case just pictured, but partial and more or less varying. In that event the deviating eye *R* would become not perfectly but only approximately aware of its true direction, so that it would get the impression that it was pointing more or less toward *G* but not actually at it. Its visual axis then would neither be regarded as having its true direction, *RG*, nor yet as being transferred to *OC*, but as having some intermediate direction. In that case, *C* would neither be projected to its true place as when the reversion to the monocular type of projection was complete, nor to *H*, as when the binocular impulse was entirely dominant, but to some point intermediate between *C* and *H*. There would thus be a diplopia, but less than the amount that would correspond to the deviation. Moreover, while the deviation remained the same, the amount of the diplopia might vary, depending on which set of impulses, convergent or conjugate, got the upper hand.

This would explain, I think, satisfactorily the conditions found in incongruous diplopia, in which the images are less far apart than they should be and the amount of the double vision changes often considerably while the deviation remains the same.

This point of view may be summed up in the statement that the difference between ordinary diplopia, suppression and incongruous diplopia lies in the fact that in the first case the deviating eye, dominated by convergence impressions, locates its visual axis in the midline, *OC;* in the second case, being wholly under the influence of conjugate impressions, it locates its visual axis where it actually is, i. e., in *RG*, and in the third case, being under the influence of both convergence and conjugate impulses, it locates its visual axis along some intermediate line. *C* is projected according to its relation with the visual line as thus located.

There is no doubt that monocular projection (projection set up by conjugate movements) is of earlier development than binocular projection, which is set up by convergence action. Incongruous diplopia and suppression in which monocular projection more or less prevails over binocular represent, then, a reversion to the primitive type.

According to this view of the case, incongruous diplopia, commonly regarded as an instance of false projection, is really less false than ordinary congruous diplopia. In suppression the projection is not false; indeed, it is just because the projection is true that suppression takes place.

Suppression thus represents the terminal stage of the process that produces an incongruous diplopia, and it is not surprising that the two conditions are often associated. Furthermore, the occurrence of antipathy to single vision in cases of incongruous diplopia may be explained by supposing that when by prisms or any other means the right-eye and left-eye images are superposed there is suppression, and hence no impulse is afforded to maintain the muscular balances which, if there were no suppression, would hold the images together. One eye then swerves slightly one way or another, and diplopia at once sets in.

In suppression, not only is the object of fixation projected to its true place and thus suppressed, but the like is true of other objects. (For as has already been shown, if the eyes projected as they do in monocular vision there is no reason why the object, *B* [fig. 1 in the previous article], should not appear single. It is only the effect of postural [convergence] sensations that makes the eyes project *B* in double images.) In suppression, according to the hypothesis here presented, being emancipated entirely from the dominance of convergence, the eyes project absolutely as they do in monocular vision. Hence the two retinal images of *B* are projected to their true place and *B* consequently appears single.

It may be urged against this theory that if suppression is due to the projection of the right-eye and the left-eye images of *A* to the same point, i. e., to the place that *A* itself occupies, there ought to be binocular single vision again when the squint is cured by operation and both foveae are directed at *A*. So one would do, doubtless, if the operation were done before suppression, i. e., the habit of monocular projection, had become firmly established. But ordinarily, when a squint is cured, the relations between the cerebral representatives of the two foveae have been long in abeyance and have in all probability completely lost the ability to work together.

This theory would account for the fact that in incongruity the diplopia is regularly less than the retinal deviation and of the same character, and that this is true at the outset even in cases in which ultimately the diplopia is greater than the deviation or is of the opposite kind (paradoxical). The rare cases in which the diplopia is paradoxical from the start may be due to some special complicating factor. This appeared in the following case.

Exotropia; Paradoxical Diplopia.—In Mitchell C., aged 18, exotropia had been noticed soon after birth, and it had remained about the same in amount ever since. He had never, except once, had spontaneous diplopia. The father had observed that when red glass was put over one eye, that eye turned in. Exotropia, alternating, showed: X 11° (Sc), S 3° (P); at times, however, there was nearly, if not quite, binocular fixation at distance, and then it showed DH 3°. With the Maddox rod, DH was 11°, and when the prism 22$^{\Delta}$ (12°), base in, was superposed, DH was 22° or so. DH was about the same in all parts of the field of fixation. There was varying and inconstant LH, not increasing particularly in any

direction of gaze. At 5 meters, DH in Er and El was about the same as in Ef; X' 10° (Sc), S' 2° (P). The convergence near point was 70 mm. or less from the base line; but on nearing this point, i. e., between 70 and 100 mm., there was esotropia of the left eye and DH. At 33 cm. in Eu and r exotropia was marked in the left eye; Eu and l, exotropia was moderate in the right eye; Ei was slightly <n and was performed with effort (nystagmoid twitching) in both eyes. With bar reading, there was at first monocular (R) vision; then either alternate or binocular vision. With the amblyoscope, there were binocular single vision and apparently stereopsis; when the eyes were converged, 25° or 30°; at the same time the object appeared small and faint (probably from accommodative spasmodic effort).

In this case the eyes evidently projected almost as they would do in monocular vision; consequently, if no other factor had been in operation, the patient should have shown a varying but slight crossed diplopia. But there was evidently an additional factor, viz., a strong convergence spasm. This was shown by the fact that although the internal recti per se were obviously weak, the patient could converge strongly (to 70 mm. or less), and on nearing this point he fell into a state of spasmodic esotropia. The postural impulses derived from this sense of excessive converging effort made him feel that the diverging eye was not deviated out as far as it actually was. Hence when the left eye was deviated out 11° so that an object straight ahead formed its image 11° to the temporal side of the fovea, monocular projection acting alone would make him project the object to its proper place, provided he had an accurate appreciation of the direction in which his eye was pointing. In that case he would get union of the double images, or rather suppression of the left one. But the undue convergence effort, exerted constantly in spurring up the weak internal rectus, made him feel that the left eye was turning in somewhat, i. e., was not deviated out as much as 11°. Hence the object that formed its image 11° to the left of the fovea was not projected straight ahead to its true position but somewhat to the right of the latter, so that there was a moderate homonymous (paradoxical) diplopia.

VISUAL FIELDS IN CHRONIC GLAUCOMA

THE EFFECT OF REDUCED ILLUMINATION *

S. B. MARLOW, M.D.
SYRACUSE, N. Y.

The degree of success of treatment, whether medical or surgical, is well known to be dependent on early diagnosis in all fields of medical endeavor. This is particularly true of the chronic forms of glaucoma and has stimulated the development of reliable methods for early diagnosis and for noting the progress of the disease. The importance of careful measurement of the field of vision in these cases has long been recognized. Chronic glaucoma is more dependent on perimetry for its understanding than perhaps any other disease, and many advances in this method of examination have been associated with its study.

The most important addition to the methods of visual field examination has been the introduction of the tangent screen and the use of small test objects for the investigation of the central portion of the field. Many different kinds of screen have been developed varying in size and shape and in the methods of recording the defects uncovered. The screen devised by Thomasson is simple, convenient and perhaps more adaptable to office and clinic use than any of the larger screens. Its chief disadvantage, however, is the limitation of its use to scotometry as it is not sufficiently large to permit mapping the field of a 1 mm. object at a distance of 2 meters. Nevertheless, it has served well the purpose for which it was devised and has probably stimulated the careful investigation of the central field, especially in the early diagnosis of glaucoma.

The use of a tangent screen at 1 or 2 meters with small-sized test objects has the effect of magnifying such defects as may be present, or, to use the language of Ferree and Rand, of increasing the sensitivity of the test. As these authors have pointed out, there are three ways in which this can be done. The first is exemplified by the present method of using the tangent screen. A second has to do with the difference between the object and its background. This factor they have incorporated in a perimeter of their own design. The third variable concerns the illumination. Combinations of these offer further possibilities.

It is my purpose in this paper to report the effect of the combination of the first and third in a small series of cases. The method used,

* Submitted for publication, Sept. 1, 1931.

although not scientifically controlled in a strict sense, seems to have definite value in spite of its crudeness. It is described because it reduces to a minimum additional apparatus and expense and can be made use of without adding greatly to the time consumed.

In all cases Thomasson's screen was used at a distance of 1 meter and a test object of 1.5 or 1 mm. After adjusting the patient before

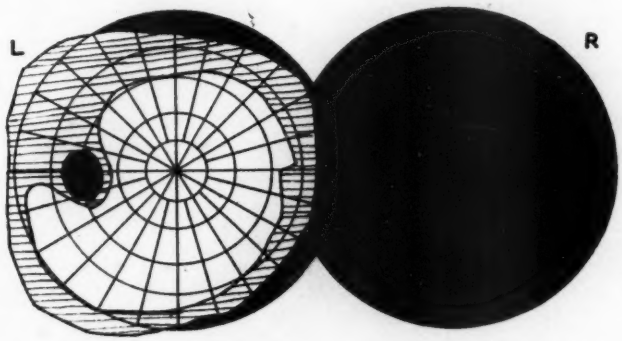


Chart 1 (case 1, Feb. 18, 1929).—Vision, 6/9; tension, 2/5.5 (29 mm.). In this and the following charts, the blackened area represents the 1.5/1,000 field with full illumination; the shaded area, loss in dim light. Note the appearance of a Ronne step.

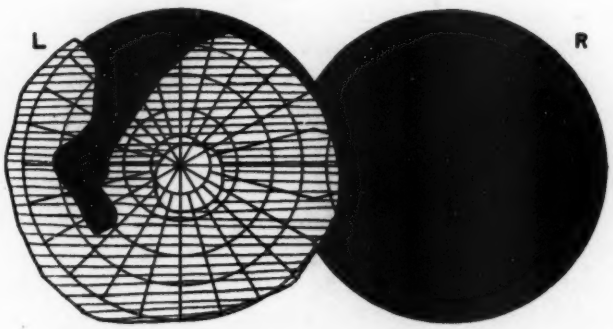


Chart 2 (case 1, Feb. 12, 1930).—Vision, 6/8; tension, 3/5.5 (25 mm.). Note the scotoma connected with the enlarged blind spot with full light and much more marked contraction of the 1.5/1,000 field in dim light.

the screen, the lighting was reduced by closing window curtains which consist of venetian blinds and heavy interlined drapes which reduced the illumination of the screen to an average of about 0.2 foot candles. After allowing for adaptation to take place, the 1/1000 field was outlined, repeated tests being made to avoid error. After this had been completed, the curtains were widely opened and the tests repeated, the blind spot being carefully mapped and the search for scotomas

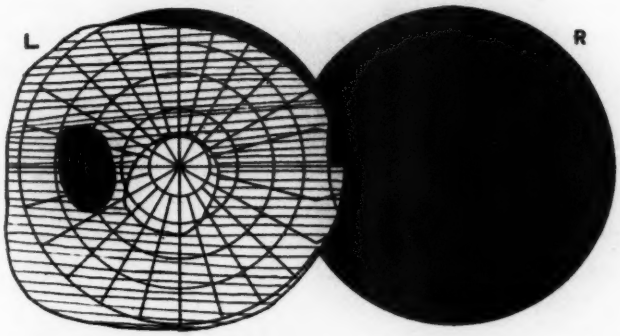


Chart 3 (case 1, July 30, 1930).—Vision, 6/8; tension, 3/5.5 (25 mm.). Except for an enlarged blind spot and nasal step in good light there is very little change.

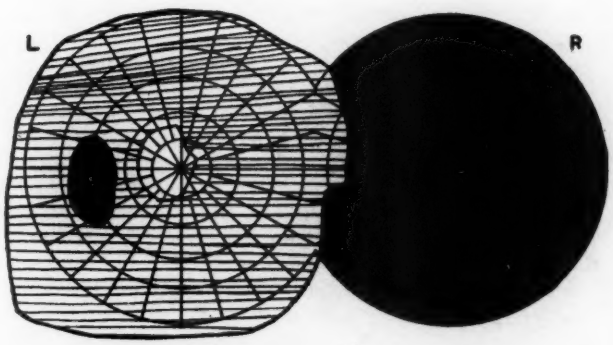


Chart 4 (case 1, Jan. 20, 1931).—Vision, 6/8; tension, 4/7.5 (31 mm.). The blind spot is enlarged vertically and the nasal step shows greater contractions of the 1/1,000 field in dim light.

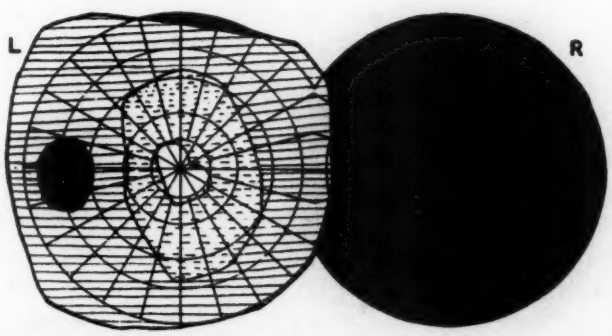


Chart 5 (case 1, Jan. 30, 1931).—Vision, 6/8; tension, 3/5.5 (25 mm.). The 2/1,000 field was plotted in dim light. Some further contraction of the 1/1,000 field is seen in dim light with the large field free from scotoma in good light.

carried out. When these tests were first carried out immediately after the patient left, a normal person was examined with reduced illumination for control.

Chronic glaucoma seems an especially appropriate disease for which this procedure can be used. The work of Derby and his collaborators has clearly demonstrated that a lowering of the light minimum is probably the earliest detectable change in incipient cases. This being true, one should be able to detect earlier or more advanced field changes in these cases by reducing the illumination. Furthermore, progress of the disease could be more surely demonstrated.

With these considerations in mind, the fields in eleven cases of chronic glaucoma watched over varying periods of time are presented.

Six charts are presented from case 1. This patient has been under observation since September, 1923, and has had repeated field studies made over a period of more than seven years. She is, therefore, well trained and an accurate observer. Many attempts to discover scotomas or other changes in the central field and about the blind spot have always been unproductive. Her right eye was blind when first seen. The first chart presents a normal blind spot and the field for a 1.5/1000 test with full illumination. The shaded area represents the reduction of the field when the light was reduced. Chart 2, taken one year later, shows the only scotoma ever found in this case connected with the blind spot. Except for this area, the 1.5/1000 field shows practically no change. With reduced illumination, there is a marked contraction of the 1.5/1000 field as compared with the previous chart. The conditions of the test are practically the same as regards the time and brightness of the day. In chart 3, the chief changes are represented by a definite enlargement of the blind spot and the nasal step. In chart 4, taken six months later, the blind spot remains enlarged, and there is little change in the 1/1000 field with good illumination. There is, however, a further reduction of the 1/1000 field with reduced lighting. Further change is noted in the fifth chart in which a 2/1000 field is plotted for the first time with reduced light. In the last chart (6), the 1 mm. object is no longer visible when the light is reduced, and the 2/1000 field has undergone still further contraction. Throughout all these observations it will be noted that there has been relatively little change in the extent of the 1/1000 field when good lighting is used. There has been some variation in the blind spot, but no scotoma has ever been discovered, with the exception noted, although a careful search has been made. The striking thing about these charts, then, is the gradual reduction of the field in a dim light without appreciable change in good light. This sequence of events corresponds well with other clinical evidence of the advance of the process. The patient has been having more subjective symptoms lately, the tension has been more

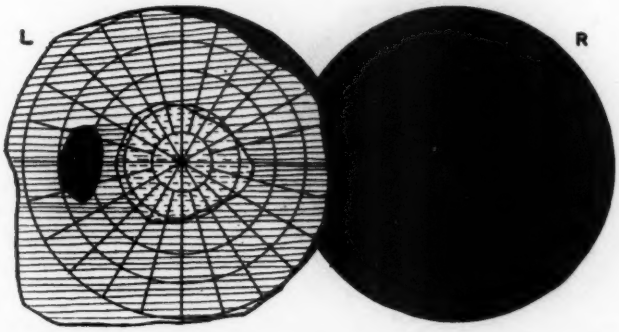


Chart 6 (case 1, Feb. 14, 1931).—Vision, 6/6—; tension, 4/5.5 (22 mm.). There is no change in the 1/1,000 field in good light. In dim light, the 1/1,000 field is lost entirely and there is further contraction of the 2/1,000 field.

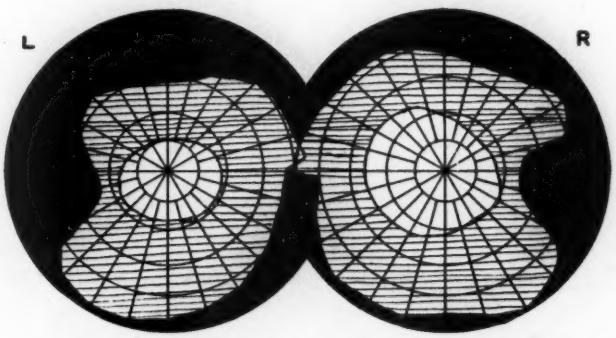


Chart 7 (case 2, March 8, 1929).—Vision, 6/6— each; tension in the right eye, 4/5.5 (22 mm.), and in the left, 5/5.5 (18 mm.). There is contraction of the 1.5/1,000 field with nasal steps in good light, with still greater loss in dull light.

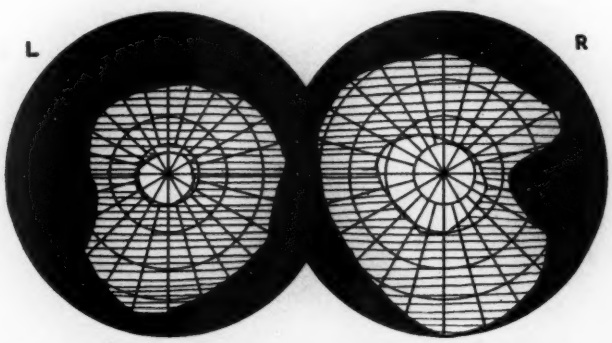


Chart 8 (case 2, May 8, 1929).—Vision, 6/6— each; tension, 20 mm. each. No change is noted except further contraction of the 1/1,000 field in dull light.

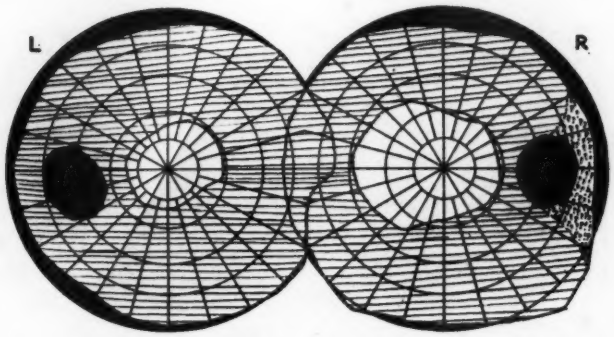


Chart 9 (case 2, Oct. 1, 1930).—Vision, 6/6— each; tension in right eye, 22 mm., and in left eye, 18 mm. With good light, there is apparent improvement; the 1/1,000 field is much enlarged. With reduced light, the 1/1,000 field is lost; the 2/1,000 field, greatly contracted.

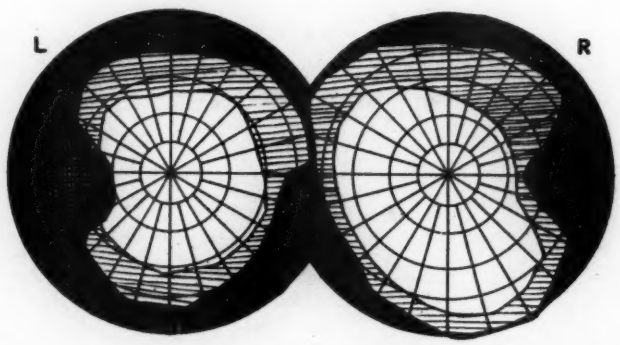


Chart 10 (case 2, Dec. 3, 1930).—The patient suffered a recent attack of pain with the observation of halos. Vision, 6/6— each; tension, 18 mm. each. There is contraction of the 1/1,000 field in good light, and complete loss of the 1/1,000 and 2/1,000 fields in dim light with contractions of the 4/1,000 field in the dim light.

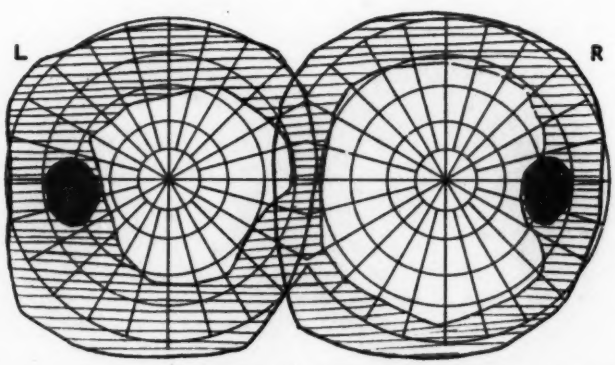


Chart 11 (case 2, Dec. 17, 1930).—Apparent improvement is shown in the 1/1,000 field in good light. With reduced illumination, there is not much change; the 3/1,000 field is contracted, and the 1/1,000 and 2/1,000 remain lost.

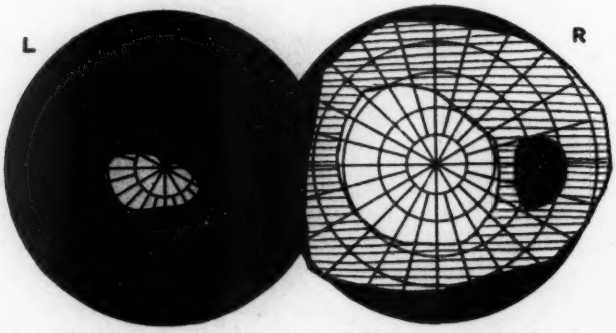


Chart 12 (case 3, May 10, 1930).—Vision, 6/6— each; tension in the right eye, 30 mm., and in the left, 35 mm. There is marked central loss of the left eye with a steep edge as indicated by coincidence of the 1.5/1,000 and 12/1,000 fields. The field of the right eye showed some peripheral loss for the 1.5/1,000 test with good illumination, and with greater contraction when the light is dull.

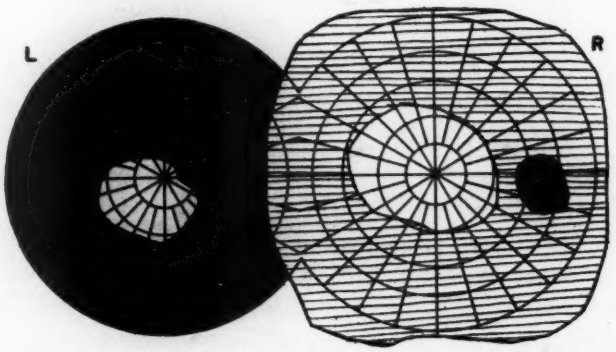


Chart 13 (case 3, Jan. 2, 1931).—Vision, 6/6— each; tension in the right eye, 3/5.5 (31 mm.), and in the left, 2/7.5 (41 mm.). The left hand field shows no appreciable change. With full illumination, the right hand field for 1/1,000 is definitely larger, but in dull light there has been some further though slight contraction of the 1/1,000 field.

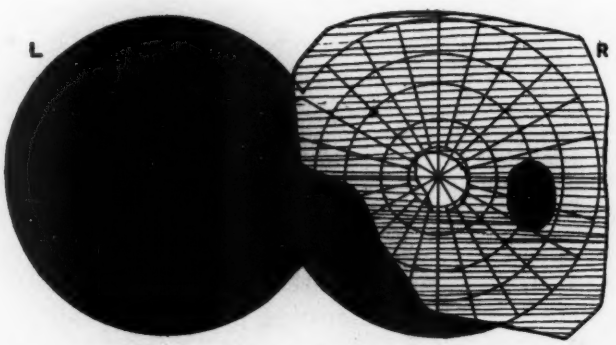


Chart 14 (case 4, March 17, 1930).—Vision, 6/6—; tension, 4/5.5 (22 mm.). The 1.5/1,000 field showed loss in the lower nasal quadrant in good light and much greater contraction in dull light.

difficult to control with miotics and some surgical intervention seems imminent in the near future in spite of the fact that the patient is a poor surgical risk.

Five charts show the progress of the disease in case 2. There is a greater degree of variation in this case, but it seems fair to conclude that the fields in reduced illumination show the progress of the disease more clearly and are more consistent with the other clinical features of the case than those taken with good light. This is not a case of primary simple glaucoma, for the slit-lamp revealed the presence of very fine deposits diffusely scattered over the posterior surface of the cornea. The change shown in charts 7 and 8 consists mainly in a slight reduction in the 1.5/1000 field when the lighting was reduced. A marked change is to be noted in chart 9. If one were to consider the fields taken with good light only, a very marked improvement would be thought to have occurred. However, the 2/1000 field is quite contracted with a reduced illumination and 1/1000 invisible. No explanation is offered for the increased size of the 1/1000 field in good light. In chart 10, still further progress is noted for at this time the 2/1000 field has become lost. Chart 9 indicates a second apparent improvement, the 1/1000 and 2/1000 field remaining lost, however. These ups and downs corresponded fairly closely with the clinical course of the disease. The patient suffered recurrent attacks of pain which were coming at more frequent intervals, although the tension was always found within normal limits. The depressions in her field were found when they were examined relatively soon after discomfort, the larger fields at a longer interval after the occurrence of subjective symptoms.

The patient in case 3 has been under observation less than one year. Charts 12 and 13 show improvement under treatment if the extent of the field and the size of blind spot alone are considered. With the light reduced, this improvement disappears, the second field showing some reduction in size from the first.

In case 4, charts 16 and 17 show some slight improvement in the field after operation. This patient has been under observation for nearly four years. Soon after he came under observation, an iridectomy was done on his right eye. Charts 14, 15 and 16 show the failure of his left eye, which was found more and more frequently to have elevated tension not fully controlled with drops. It is interesting to note the reduplication of the defects found with full illumination intensified by dim light.

The patients in the remaining cases have been observed for varying periods, but only one field examination under the conditions stated is available, either because the patient has not returned for further observation or because the case is too recent.

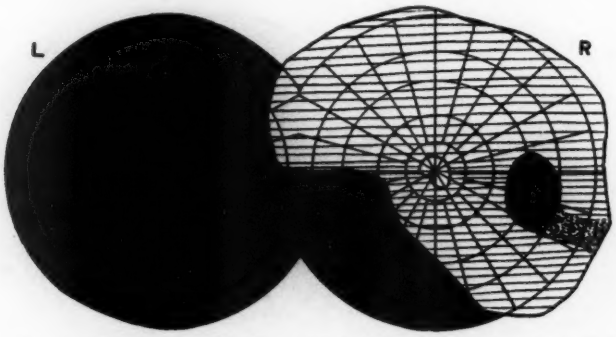


Chart 15 (case 4, May 29, 1930).—Vision, 6/8—; tension, 6/7.5 (23 mm.). With good light, there was some additional loss in the nasal field, but a much greater loss with the light reduced.

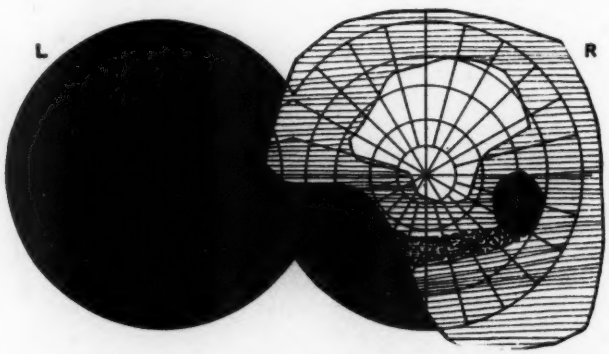


Chart 16 (case 4, Sept. 30, 1930).—Vision, 6/8+; tension, 3/7.5 (34 mm.). There is no change in the 1/1,000 field with full illumination and complete loss of the 1/1,000 field in dull light with marked contraction of the 2/1,000 field. There is also an indefinite band of relative scotoma extending from the blind spot to the nasal field.

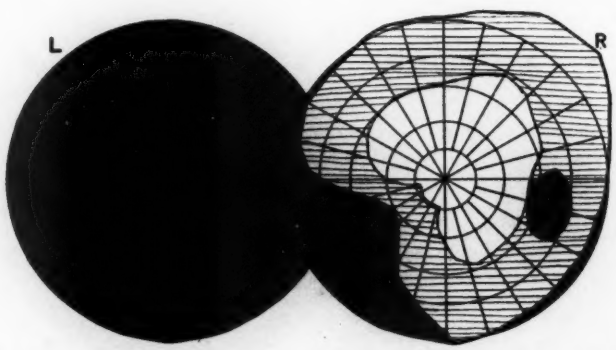


Chart 17 (case 4, March 7, 1931).—Vision, 6/10; tension, 5/5.5 (18 mm.). There is no change in the 1/1,000 field with good light; the 2/1,000 field is somewhat larger in dull light, the 1/1,000 field remains lost. Diminution of vision due to lens changes may account for this.

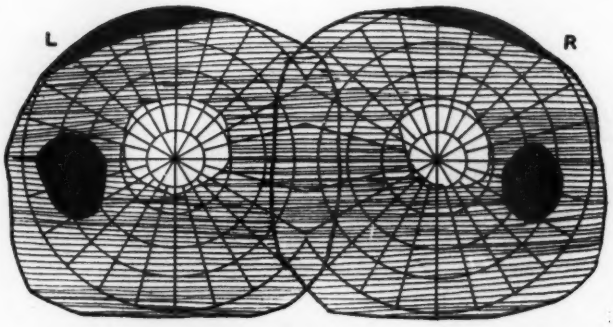


Chart 18 (case 5, June 4, 1929).—Vision, 6/6+ each; tension, 3/7.5 (37 mm.) each. The enlarged blind spots are the only change in good light. There is marked contraction of the 1.5/1,000 in dim light.

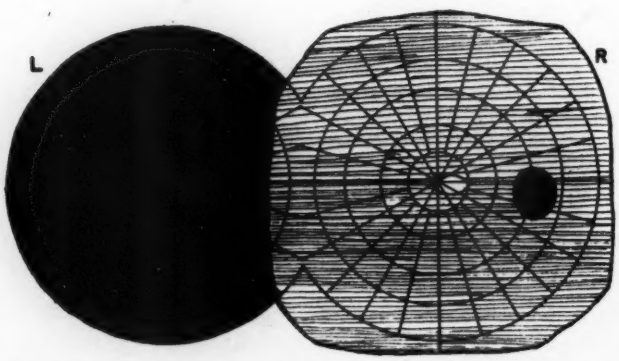


Chart 19 (case 6, Aug. 1, 1930).—Vision, 6/6—; tension, normal to the fingers. There are no changes in good light, but marked contraction of the 1.5/1,000 field in dull light.

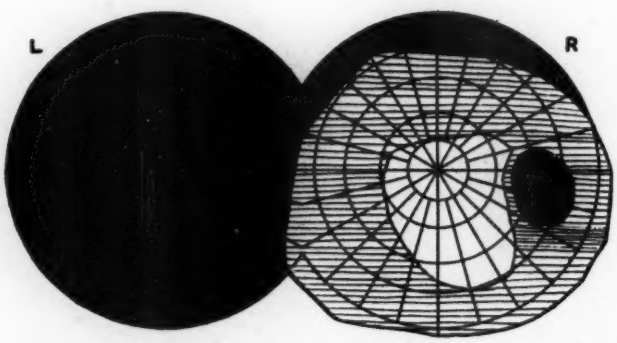


Chart 20 (case 7, June 10, 1931).—Vision, 6/12+; tension, 2.5/10 (58 mm.). With good light there is some loss of the 1.5/1,000 field above and a somewhat enlarged blind spot. In dull light, the 1.5/1,000 field is contracted.

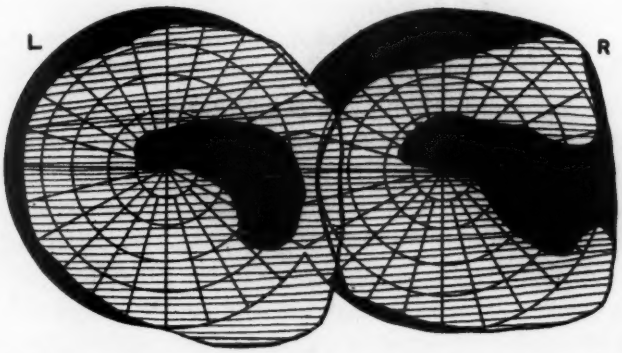


Chart 21 (case 8, June 17, 1930).—Vision, 6/12+; tension, 6/7.5 (22 mm.). The left field for 1.5/1,000 is somewhat contracted and presents a large Bjerrum scotoma in good light. The right field indicates complete loss of 1.5/1,000 up to 5/1,000 fields and shows the scotoma to be connected with the periphery.

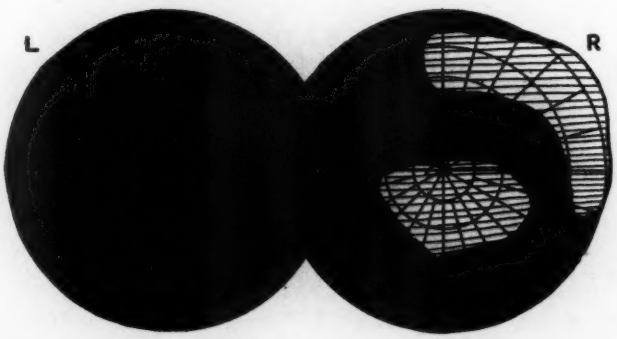


Chart 22 (case 9).—Vision, 6/10; tension, 32 mm. There is marked contraction of the 1.5/1,000 field and arcuate scotoma with good light. With reduced light, the 1.5/1,000 field is lost with extreme concentric contraction of the 3/1,000 field.

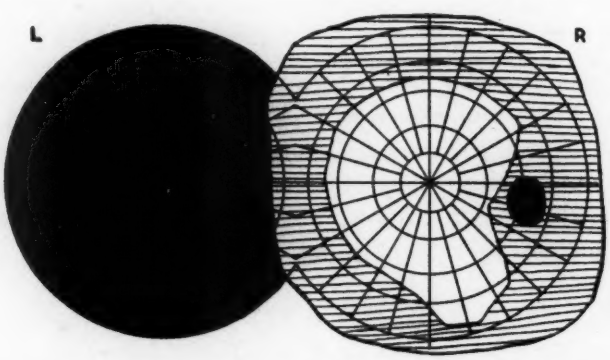


Chart 23 (case 10, June 2, 1930).—Vision, 6/10; tension, 3/7.5. There is no change in the 1.5/1,000 field in good light but definite loss in reduced.

The patient in case 5 was under observation six months only and has not been seen since operation was advised. Chart 18 shows the enlargement of her blind spots, the only changes to be found, the extent of the field being the limits of the screen except above and normal on the perimeter, with full illumination and a contraction more in accordance with the progress of the disease with reduced illumination.

The patient in case 6 has been under observation seven years. His left eye failed in spite of treatment with miotics, operation being refused. Under miotics, the disease has appeared to be controlled, although there have been occasional elevations of tension. Chart 19 shows a marked loss of his field when the light is reduced. This corresponds fairly well with his symptoms, for although his vision remains within normal limits it has fallen from 6/5 to 6/6—. He complains at each periodic visit that he does not see as well as formerly.

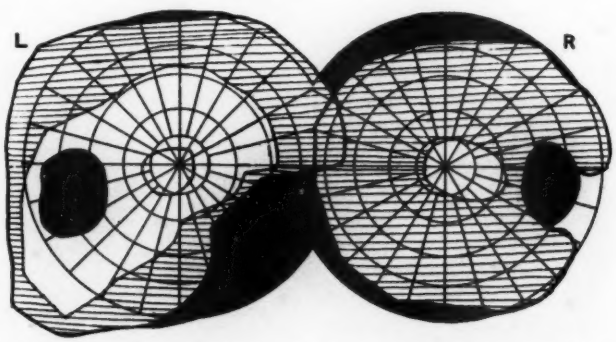


Chart 24 (case 11, Feb. 4, 1931).—Vision in the right eye, 6/18—; in the left eye, 6/18+; tension, 25 mm. each. The right field for 1/1,000 test in good light shows some peripheral contraction which includes the blind spot. The blind spot for the 3/1,000 field is enlarged. In dim light, the 1/1,000 field is lost and there is marked contraction of the 2/1,000 field. The left field for 1/1,000 in good light shows a nasal step with loss of the lower nasal field and an enlarged blind spot. In dull light, there is contraction of the 2/1,000 field, analogous to the 1/1,000 field in good light, and marked concentric contraction of the 1/1,000 field.

The patient in case 7 had a high tension but no pain. Miotics controlled the tension while he was under observation, a short time only.

Chart 21 in case 8 shows the connection of the scotoma found with full illumination with the peripheral field when the light is reduced and a larger test object is necessary. This patient was seen but once, for a second opinion, operation having already been advised by another ophthalmologist.

Cases 9 and 10 (charts 22 and 23) are clinic cases, both patients having been operated since these observations were made. The condition in case 9 was at an advanced stage, and that in case 10 in an early stage, although one eye had already been lost.

The patient in case 11 (chart 24) has been examined once. The more advanced stage of the disease in the right eye is more clearly evident in the field with reduced illumination. Although the tension was not found to be elevated at any time, both eyes improved after the use of miotics, the improvement in the left being the greater.

These observations are too few and extended over too short a period of time to allow conclusive deductions. While it is true that the procedure is crude and open to many criticisms, it is simple and may lead to earlier diagnosis than the usual method of scotometry. It should, however, be used with caution, only after the individual worker has determined for himself the limits of the normal field with the illumination reduced under his own particular working conditions or by making a comparison at the time of testing with a normal for control. In those cases in which repeated examinations have been made the results appear to have corresponded closely to other clinical features of the case under observation.

DISCUSSION

DR. LUTHER C. PETER, Philadelphia: (Dr. Peter suggested that reduction of the light sense in glaucoma would explain the field defects found. He emphasized the value of any additional method that will increase the accuracy of field determinations. He believed that a careful study of fields on the Ferree-Rand perimeter and the Lloyd slate with very small test objects will reveal the same defects under conditions that can be better controlled and are better understood. He brought out the value of small colored objects, especially green, and of following the course of the nerve fibers in detecting scotomas due to glaucoma.)

DR. MARK J. SCHOENBERG, New York: (Dr. Schoenberg reviewed some of the provocative tests for early glaucoma, such as those with caffeine, compression of the jugular, and stay in the dark room. He advised a study of 100 normal fields in reduced illumination and fields in other pathologic conditions, and suggested the use of dark glasses as a means of reducing illumination.)

CYSTS OF THE CORNEA*

MILTON L. BERLINER, M.D.
NEW YORK

In going over the literature of cysts of the cornea, one is struck by the fact that so few cases have been reported. In all there have been about fifteen cases described. In searching through large collections of pathologic preparations, I was able to find fourteen additional examples of corneal cysts.

This paper is limited to the consideration of only true corneal implantation cysts—that is, those cysts having a definite lining membrane—and does not include congenital cysts or the so-called dilated corneal lymph spaces.

From a study of the cases previously reported and from those presented here, it will be seen that these cysts fall into definite classes.

CLASSIFICATION OF CYSTS

A. *According to Location.*—1. Precorneal Cysts: There are two types of these cysts; one develops on the surface of the cornea, and the other just below the epithelium, that is, between the epithelium and Bowman's membrane. Those cysts that occur on the surface of the cornea develop where large cicatrices have resulted, or where the conjunctiva has been drawn over and become adherent to the cornea. Dr. Fuchs[1] described such a cyst occurring under a pterygium. In several cases this type of cyst developed after an attempt to excise staphylomas. In one case it occurred after an ulcer of the cornea, which was covered by an adherent conjunctiva. This type of cyst is generally lined by either surface or bulbar epithelium, and generally occurs at the limbus. In considering the etiology of these precorneal cysts that occur on the surface of the cornea, the following possibilities may account for their formation: (*a*) the property of the transitional epithelium of the limbus to proliferate; (*b*) the invaginations of the epithelial surfaces at the time of suturing (after removal of a staphyloma), and (*c*) ingrowing of the epithelium during healing of a wound.

The second type of precorneal cysts, that is, those that develop between the epithelium and Bowman's membrane, are very rare. I have been able to find only one example. This type is formed after

* Submitted for publication, Aug. 10, 1931.

* Presented at a demonstration before the New York Academy of Medicine, Section of Ophthalmology, May 18, 1931.

1. Fuchs, E.: Arch. f. Ophth. **38**:32, 1892.

an implantation of epithelial cells between the epithelium and Bowman's membrane which occurs after a detachment of the epithelium.

2. Intracorneal Cysts: These cysts are situated in the corneal parenchyma proper and are found lying anywhere in the thickness of the cornea between Bowman's membrane anteriorly and Descemet's membrane posteriorly. The type that occurs in the cornea proper is generally found in scar tissue resulting either from a perforating ulcer or injury, or after operative intervention. Owing to the compactness of the corneal tissue, they rarely attain any great size, and are practically never diagnosed during life. There are two facts to consider in their formation: a column of epithelium forming between the lips of a slowly healing wound may form a cyst either (1) by degeneration or liquefaction of the cells in the center or (2) by a direct implantation of epithelial cells into the deeper layers of the cornea with no continuous epithelial connection with the surface. These cysts are generally lined by surface epithelium. Claiborne [2] reported an intracorneal cyst which was lined by pigmented iris epithelium. In several cases, particularly when the cysts occurred near the limbus, they have extended into the sclera. It seems that after a cyst leaves the cornea and extends into the sclera, it grows to considerable size. This may be due to the fact that after the cyst leaves the cornea, it finds a line of cleavage between the scleral bundles and separates them very readily. These cysts enlarge to great size and are usually diagnosed during life. Dr. Samuels [3] reported a case in 1913 in which the cyst occurred after the removal of a staphyloma and extended posteriorly to the optic nerve, having invaded the entire space of Tenon. The large cysts that extend posteriorly and envelop the globe are called peribulbar. They are generally lined by either surface or bulbar epithelium, and have a fluid content.

3. Postcorneal Cysts: These cysts are the rarest of all types, there having been up to the present time only one case reported. They are situated in the posterior part of the cornea and are formed by Descemet's membrane and the endothelium. They are not to be confused with the anterior chamber cysts which are always lined by surface epithelium. What really happens is that there is a splitting of Descemet's membrane which later becomes lined by endothelium. It is evident that after ruptures or separations of Descemet's membrane, the free ends retract and curl up, allowing the endothelium to grow around the edges. The gap is then filled in by a new elastic membrane which is formed from the neighboring endothelial cells. In his experiments with kerato-

---

2. Claiborne, J. H.: Epithelial Corneal Cyst, Tr. Am. Ophth. Soc. **10**:588, 1903-1905.

3. Samuels, B.: Arch. Ophth. **42**:12 (June) 1913.

plasty, Mr. Thomas [4] showed that even very large gaps in Descemet's membrane were soon filled in by a new elastic membrane. Through the courtesy of Dr. Fuchs, I examined sections of the eye from the only known case of posterior corneal cysts which was reported by Tertsch.[5] He believed that due to distention by edema the corneal lamellae either became displaced or ruptured, and that consequently the more elastic and resistant Descemet's membrane became detached. Later, the prolonged pressure combined with increased corneal protrusion finally broke down the strength of Descemet's membrane and produced a tear. The endothelium was then able to grow around the torn edges and coat not only the free Descemet's membrane but also the posterior corneal surface, which would form the anterior wall of the cyst.

B. *According to Lining of Cyst.*—1. Epithelial: There are two types of epithelial lining:

(*a*) Corneal surface epithelium. Excepting for the postcorneal cysts, a majority of the cysts were lined by this type of epithelium.

(*b*) Bulbar or conjunctival epithelium. Some of the precorneal and the corneoscleral cysts were lined by bulbar epithelium.

2. Endothelial: This type is found chiefly in postcorneal cysts. Feleti reported a case of intracorneal cyst lined with endothelium. This cyst had no communication with the anterior chamber. He ascribed the presence of endothelium to a transformation of the fixed cells of the cornea. This explanation seems to be rather unsatisfactory. It is possible that a single layer of surface epithelium compressed by intracorneal tension may resemble endothelium.

3. Pigment Epithelium of the Iris: A cyst lined with pigment epithelium of the iris must of necessity have been in communication with the anterior chamber at some time. In these cases the iris is always found in contact with the posterior surface of the cornea and is generally very atrophic. The pigment cells proliferate and line the cavity of the cyst. This is what occurred in Claiborne's case. I have also been able to find a case of this sort.

4. Of Uncertain Origin: In some cases it was extremely difficult to identify the type of cells forming the lining.

C. *According to Conformation.*—1. Monolocular: These cysts have one cavity. Most of the intracorneal and postcorneal cysts are of this variety.

2. Multilocular: These cysts have more than one cavity. This type was seen in several instances in cases of large corneoscleral cysts.

---

4. Thomas, J. W. Tudor: An Experiment in Keratoplasty, Proc. Roy. Soc. Med. **23**:75 (Aug.) 1930.

5. Tertsch, R.: Eine Cyste an der Hornhauthinterfläche, Arch. f. Ophth. **56**: 303, 1903.

REPORT OF CASES

Precorneal Cysts.—CASE 1.—A boy, aged 14 years, had a staphyloma of the cornea. At the age of 4 the right eye was operated on for excision of the staphyloma. Several years later the eye became inflamed, hard and painful, and was enucleated.

On pathologic examination sections showed an atrophic globe with total detachment of the retina. The anterior part of the eyeball was occupied by a projection extending from the sclera below over the cornea. This projection on the surface of the cornea had been taken clinically again to be a reformed staphyloma, but microscopically, this proved not to be the case. The site of the cornea was occupied by a cicatrix which formed the posterior wall of a large cyst. This cyst was seated on the sclera below and extended over the corneal cicatrix. At its base it measured 600 microns in length. Although collapsed, it measured 400 microns across. Its inner surface was lined by a laminated pavement epithelium, and in some places by well developed basal cells.

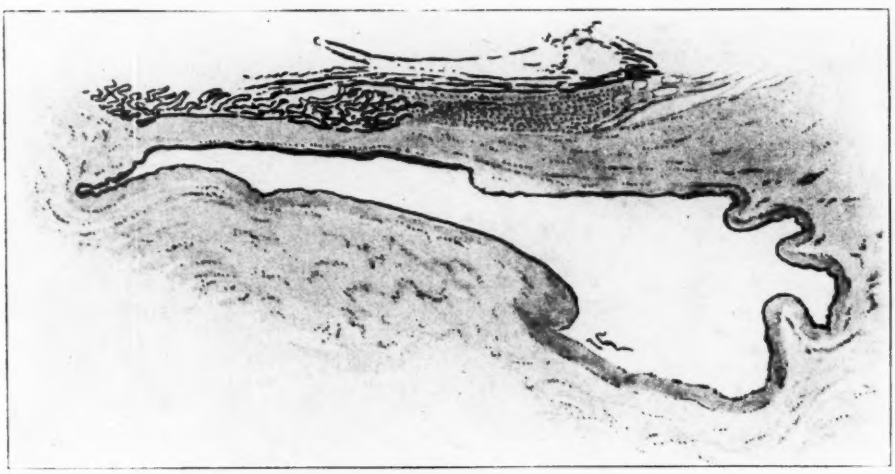


Fig. 1 (case 1).—Precorneal epithelial cyst.

This case demonstrates a monolocular precorneal epithelial cyst which was formed by an invagination of surface epithelium into the cicatrix.

CASE 2.—A man, aged 40, had a foreign body embedded in the region of the upper limbus. The foreign body was removed. After several days he returned with a well developed ulcer in this region. The ulcer then perforated the cornea. The anterior chamber became filled with exudate, and endophthalmitis resulted. Later, the eye was enucleated.

Pathologic examination revealed the whole cornea to be covered by a vascular membrane due to the fact that the conjunctiva was pulled over it. This part of the conjunctiva was of varying thickness, so that two higher projections were formed. One of these contained a cyst. This cyst was situated toward the nasal side of the cornea and measured 600 by 400 microns. It was lined on its posterior wall by a regular laminated pavement epithelium, while the anterior wall was composed only of a double layer of flat cells. From the site of the cyst directed

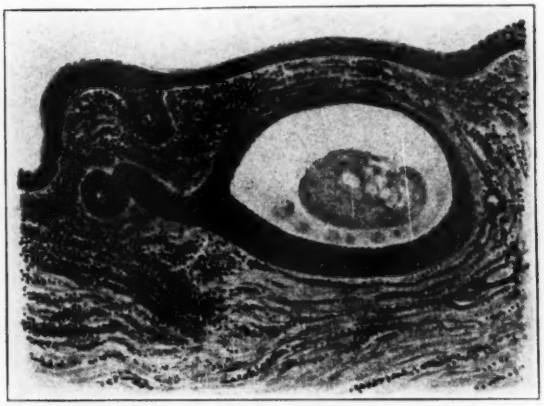


Fig. 2 (case 2).—Precorneal epithelial cyst.

Fig. 3 (case 3).—Precorneal cyst.

toward the center of the cornea, there projected a solid strand of epithelial cells. Anteriorly, there was an invagination of the surface epithelium which, however, in no place connected with the more deeply seated strand.

This case represents a monolocular precorneal epithelial cyst. The epithelium forming the cyst was either corneal or bulbar, and hence should be classified as of uncertain origin.

CASE 3.—A boy, aged 10, was struck in the right eye with a sharp pointed dart. The point of the dart perforated the cornea temporally near the limbus and entered the lens. The anterior chamber was shallow and was filled with soft lens matter. The eye became very inflamed, hard and blind, and was enucleated.

Pathologic examination revealed a perforating wound extending through the thickness of the cornea into the anterior chamber. The wound was filled with granulation tissue and lens capsule. The surface epithelium extended over the edges of the gaping wound and lined its lateral walls, for one third its depth. On the surface of the inner lip there was an epithelial cyst almost completely filled with flat poorly staining epithelial cells.

This case represents a monolocular precorneal cyst which was formed by an invagination of the surface epithelium into the lips of a wound.

CASE 4.—A blacksmith, aged 52, was struck in the left eye by a piece of iron. There was a perforating wound at the corneoscleral junction above. The eye became very painful and was removed five days later.

Pathologic examination showed between the epithelium and Bowman's membrane a lentiform body composed of epithelial cells. This body had an anterior convex surface and a plane posterior surface resembling a planoconvex lens. The epithelium covering the anterior surface of this body, due to compression from behind, was much thinner, being composed of three or four layers of flat cells without any cylindric or basal cells. Nowhere in the vicinity of the cyst was there any traces of inflammation. The cyst itself was distinguished by the fact that it was composed exclusively of epithelium without any basement membrane, and that the only connective tissue present was found at the periphery of the cyst. The epithelial wall of the cyst in its posterior part was seated on Bowman's membrane and was twice as thick as the anterior wall. The inner surface of the cyst was smooth and sharply outlined. The contents consisted of spherical and polyhedral cells which exhibited indistinct traces of nuclei. These cells are probably to be considered as cast-off smaller epithelial cells.

One must assume here that the injury caused a defect in the epithelium and that this was soon filled in by a proliferation of adjacent cells. This proliferation went beyond the necessary limits so that not only was the gap filled, but an excess of cells also penetrated under the epithelium. There they proliferated, while their connection with the surface disappeared. It must be remembered that this whole process took place in an extremely short time as the eye was removed five days after the injury.

This case represents a precorneal cyst of the second type; i. e., one situated between the epithelium and Bowman's membrane.

Intracorneal Cysts.—CASE 5.—A man, aged 35, sustained an injury to the left eye by a small gunshot. The wound failed to heal properly and was somewhat retracted. The eye was inflamed and soft. Four weeks later it was enucleated.

Pathologic examination showed a perforating wound in the sclera, temporally and below. In the cornea, above, there was another wound extending into the middle layers. Here there was an oval epithelial cyst measuring 200 by 250 microns. The wall of the cyst was composed of five or six superimposed layers of epithelial cells, the inner surface being smooth. The contents of the cyst consisted of a red-staining mass, in which, besides a few pale nuclei, there was a mass

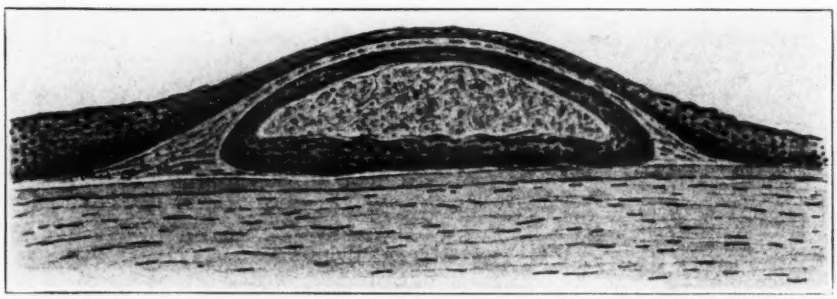


Fig. 4 (case 4).—Precorneal cyst situated between the epithelium and Bowman's membrane.

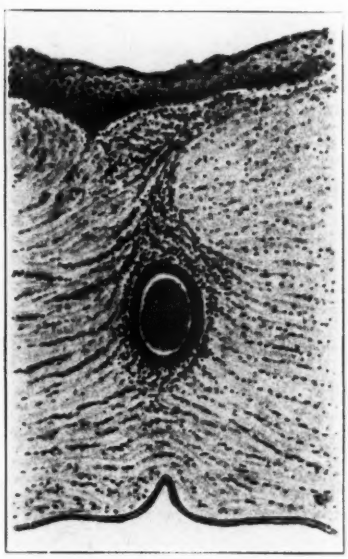


Fig. 5 (case 5).—Intracorneal epithelial implantation cyst.

of deeply blue-staining irregular bodies, probably pyknotic nuclei. The mass almost completely filled the cyst. The cornea exhibited a slight infiltration with leukocytes, and in its central part there was a disappearance of nuclei indicating a necrosis.

In this case there was an implantation epithelial cyst in the middle layers of the cornea. There is no visible connection between the cyst and the surface epithelium.

CASE 6.—The patient, aged 71, developed iridocyclitis after a cataract extraction in the right eye. Four days after the extraction, the eye was markedly inflamed and there was heavy exudate in the anterior chamber. There was no light perception. The eye was painful, and the scar was retracted. An enucleation was advised.

On pathologic examination sections showed a flattened globe with scar at the limbus above running diagonally through the cornea. Situated in the scar in about the middle of the corneal thickness was a small circular epithelial cyst measuring 150 microns in diameter. Its wall was made up of laminated pavement epithelium of about six cells in thickness. The cavity of the cyst was filled with flat epithelial cells.

This is an intracorneal cyst formed by an implantation of surface epithelium.

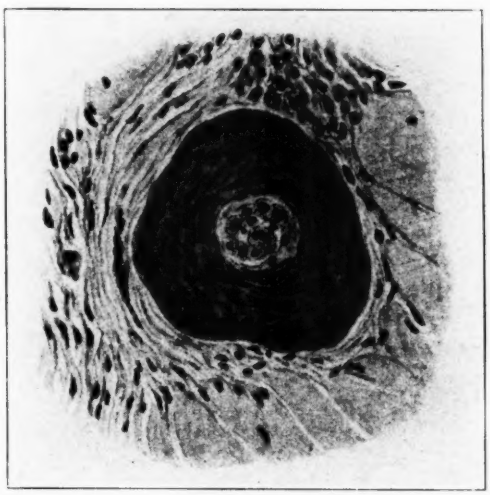


Fig. 6 (case 6).—Intracorneal epithelial implantation cyst.

CASE 7.—K. B., a man, aged 50, fourteen days before coming to the hospital was struck by a splinter of wood in the right eye. There was a perforating wound with prolapsed iris near the limbus nasally. After two weeks the eye was very congested and painful. Enucleation was then performed.

Pathologic examination showed nasally in the cornea a perforating wound as indicated by a connective tissue pathway through its thickness. This connective tissue extended into the anterior chamber. In the middle of the scar there was a small circular epithelial cyst measuring 50 mm. in diameter. Its wall was formed by a four layer thickness of epithelial cells. There was an indication of a small cavity formation in the center which was partially filled by a few flat epithelial cells.

This is an intracorneal epithelial implantation cyst and demonstrates the transition stage from a solid form of epithelial implantation to that of a cyst formation.

Case 8.—J. G., a man, aged 38, was injured in the right eye by a knife. The eye was blind. The globe was atrophied, and the cornea showed a small scar above. The scar was highly vascularized. Several months later the eye was enucleated.

Pathologic examination showed in the anterior one third of the cornea a long spindle-shaped epithelial cyst measuring 900 by 100 microns in width. Its anterior and posterior walls were very narrow and were composed of a two cell layer of flat epithelial cells. At each end, the epithelial wall was elongated and connected

Fig. 7 (case 7).—Intracorneal epithelial implantation cyst demonstrating transition stage from the solid form of epithelial implantation to that of cyst formation.

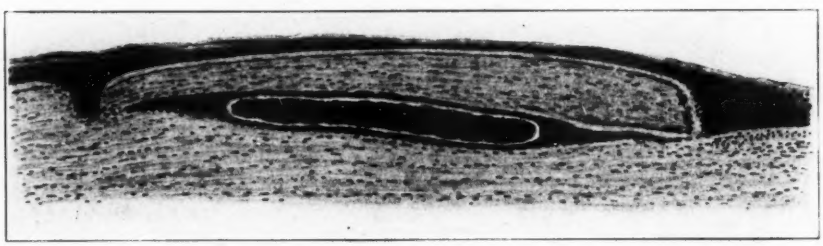


Fig. 8 (case 8).—Intracorneal epithelial implantation cyst demonstrating a cyst having an actual connection with the surface epithelium.

with an invaginated process from the surface epithelium. On one side Bowman's membrane was reflected over the anterior surface of the cyst for a short distance. The cyst contained numerous flat epithelial cells.

This is an intracorneal epithelial implantation cyst. It is rare because of the fact that it represents a cyst having an actual connection with the surface epithelium.

Case 9.—A woman, aged 40, developed a severe iridocyclitis after a cataract operation in the left eye. This was followed by secondary glaucoma. The eye was subsequently removed.

Pathologic examination showed a scar at the limbus running obliquely through the corneal thickness. Just below the surface of the cornea and in the scar was a spindle-shaped epithelial inclusion, having only a very small cavity in its center. The cavity of the cyst was filled with a few flat epithelial cells. There was no connection with the surface epithelium.

This is an intracorneal cyst showing only a small central area of liquefied epithelial cells.

CASE 10.—The patient developed an iridocyclitis in the right eye, which was followed by a complicated cataract and glaucoma. The eye was removed because of the possibility of intra-ocular tumor.

Pathologic examination showed in the deeper layers of the cornea an elongated cystic space which was almost completely lined by iris-pigmented epithelium. The

Fig. 9 (case 9).—Intracorneal cyst showing only a small central area of liquefied epithelial cells.

iris was adherent to the posterior corneal surface. It was difficult to explain the presence of this pigment lining excepting on the basis that at some time there must have been a connection with the anterior chamber which permitted the pigment to migrate into the cornea and line the cyst. In the sections available there was no communication found between the cyst and the anterior chamber.

This case represents an intracorneal cyst which was lined by iris pigmented epithelium.

CASE 11.—A youth, aged 17, came to the clinic with an uncertain history of having had trouble with his right eye since childhood. The cornea was almost completely opaque and above at the limbus a large cystic protrusion was seen.

Pathologic examination showed at the limbus an intracorneal cyst which had extended into the layers of the sclera where it had enlarged to a great size. This cyst was lined by flat pavement epithelium and was empty.

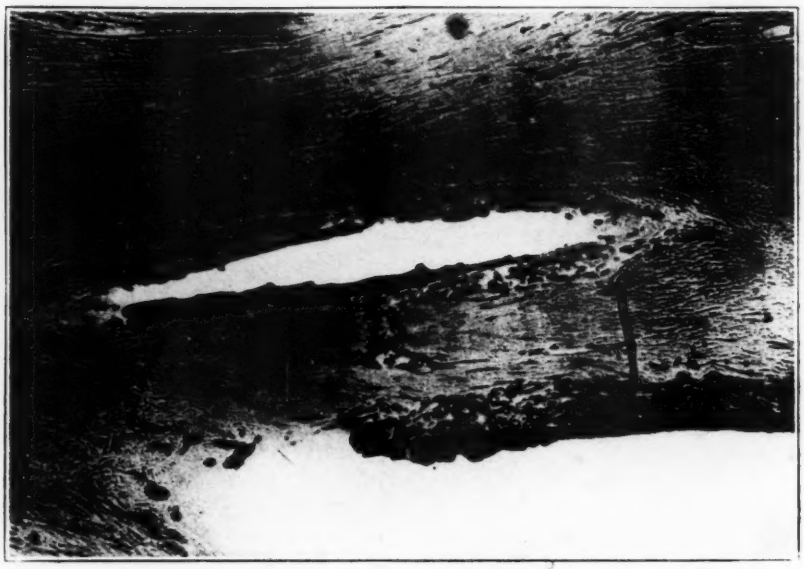


Fig. 10 (case 10).—Intracorneal cyst lined by iris pigmented epithelium.

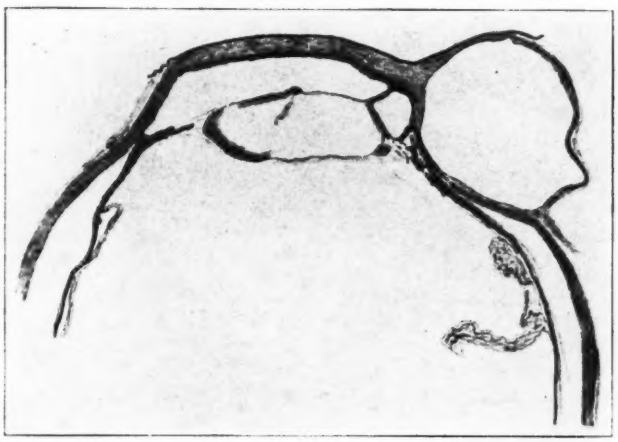


Fig. 11 (case 11).— Intracorneal cyst of the corneoscleral variety.

This is a case of an intracorneal cyst of the corneal-scleral variety, and demonstrates that cysts that leave the cornea and migrate into the sclera attain great size.

CASE 12.—A man, aged 60, had a cataract extracted from the left eye. The eye was highly myopic. Considerable vitreous was lost at the time of operation. Later, it was seen that the lips of the wound were gaping. The eye quieted down, and about six months later there was a large cystic protrusion at the site of the wound. This large cyst extended backward and filled out Tenon's space above. It had a fluid content. There was a complete detachment of the retina, and the eye was removed.

Pathologic examination revealed a large cyst which extended posteriorly from the site of the wound almost to the optic nerve. The walls of the cyst were composed of stretched scleral fibers which were lined by flat epithelial cells.

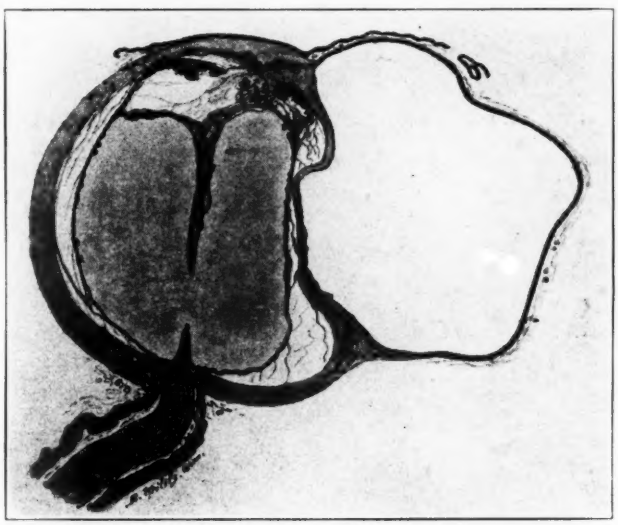


Fig. 12 (case 12).—Peribulbar corneoscleral implantation cyst.

This case represents a large peribulbar corneoscleral implantation cyst which was probably caused by an invagination of the bulbar epithelium into an operative wound.

CASE 13.—Two years after an iridectomy for absolute glaucoma, the patient returned to the hospital for examination. There was a large circular mass 10 mm. in diameter, extending from the horizontal meridian on the temporal side around the cornea to the vertical meridian below.

Pathologic examination showed a large cyst which had been formed in the middle layers of the cornea and had extended backward into the sclerotic layers. The walls of the cyst were formed by compressed corneal and scleral fibers, and were lined by a thin layer of epithelium. It had several connecting cavities. In addition to serum within the cavity, there was some blood adherent to the wall.

Fig. 13 (case 13).—Multilocular corneoscleral cyst.

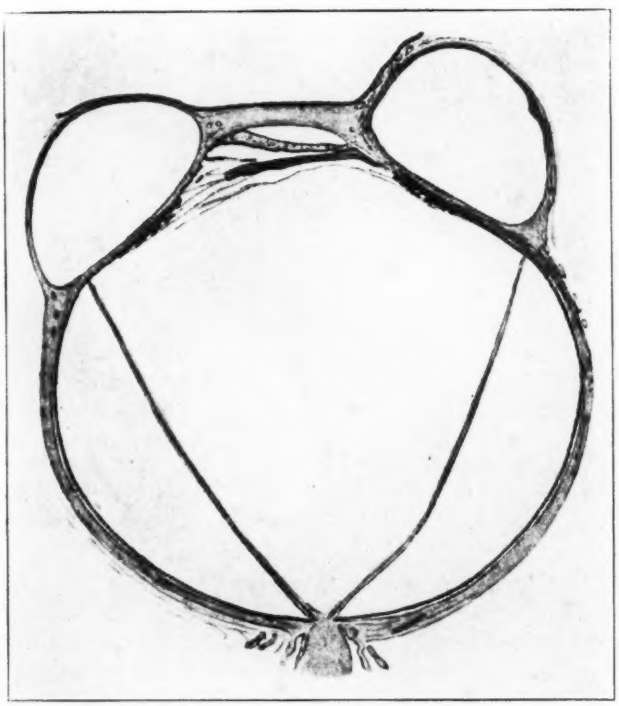


Fig. 14 (case 13).—Cyst extending around the cornea.

This represents a large peribulbar corneoscleral cyst which partially encircled the cornea and was multilocular in type.

CASE 14.—This case was reported by Dr. Samuels in 1913 from the clinic of Professor Elschnig. A man, aged 30, developed an ulcer after the removal of a foreign body from the cornea of the right eye. The ulcer perforated, and a staphyloma formed which was excised. Three years later the patient returned with a large cyst formation. The cyst protruded between the lids, and no eyeball was to be seen. A small incision was made into the tough outer coat of the cyst

Fig. 15 (case 14).—Peribulbar implantation cyst entirely encircling a shrunken globe. (After Samuels.)

liberating a clear fluid. By enlarging the incision, the shrunken eyeball came into view as it lay in the bottom of the cyst. The whole cyst and eyeball was then dissected out. This cyst was lined with a coating of stratified epithelium.

This case demonstrates a large peribulbar implantation cyst that entirely encircled a shrunken globe. The cyst filled out Tenon's space as far back as the optic nerve, and I believe it is the largest on record.

Postcorneal Cysts.—CASE 15.—Through the courtesy of Dr. E. Fuchs, I am able to show sections of the case reported by Tertsch. As stated before, this is the only case of postcorneal cyst that has been reported. This case occurred in

a young girl, aged 12, who from birth had an enlarged eye. In the cornea there was a transverse ribbon measuring 0.5 micron in width which was more densely infiltrated than the surrounding cornea. The eye was hard. Serial sections showed that Descemet's membrane was detached and stretched as a free cord across the upper posterior portion of the cornea, thereby forming a cyst. Above, there was a tear permitting free communication with the anterior chamber. Tertsch believed

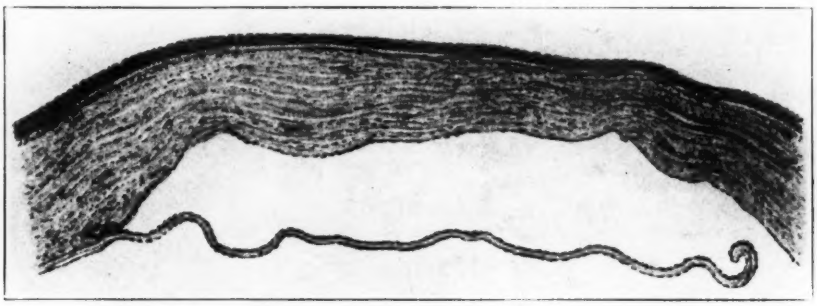


Fig. 16 (case 15).—Postcorneal cyst. (After Tertsch.)

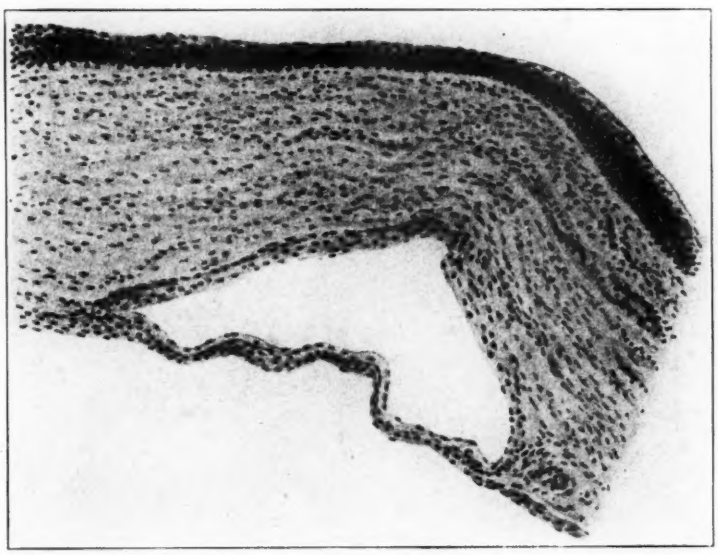


Fig. 17 (case 15).—Section through the lower part of a postcorneal cyst. (After Tertsch.)

that due to distention by edema the corneal lamellae either became displaced or ruptured, and that consequently the more elastic and resistant Descemet's membrane became detached. Later, prolonged pressure combined with increased corneal protrusion finally broke down the strength of the membranous tissue and produced a tear at the upper insertion. The endothelium was then able to grow around the torn edge and coat not only the free Descemet's membrane, but also the posterior corneal surface, which in turn formed the anterior wall of the cyst.

CASE 16.—I have been able to find only one other case that could be classified as a postcorneal cyst. This occurred in a man 48 years of age. The right eye was aphacic. Operation had been performed for extraction of a cataract several years before. Three weeks before admission to the hospital, the patient developed a serpiginous ulcer of the cornea in this eye. The eye was very congested and painful. There was no light perception, and enucleation was advised.

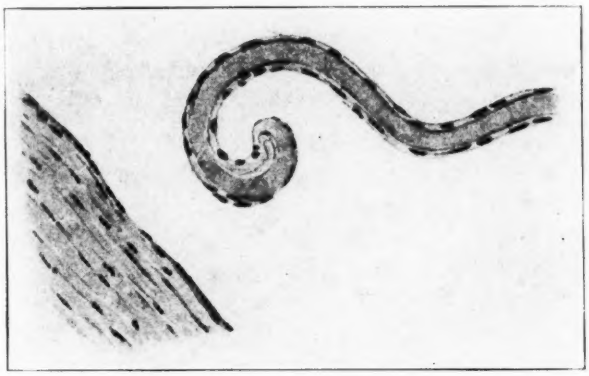


Fig. 18 (case 15).—Specimen demonstrating the manner in which endothelium grows around the torn edge of Descemet's membrane.

Fig. 19 (case 16).—Postcorneal cyst showing inclusion of Descemet's membrane with its endothelial lining.

Pathologic examination showed in the center of the cornea and on its posterior surface an endothelial cyst measuring 160 by 60 microns. The inner wall was lined by a single layer of cuboidal endothelial cells, corresponding to the normal endothelial lining of a posterior corneal surface. The outer wall, that is, toward the anterior chamber, was formed by a double layer of flatter endothelial cells, with its newly formed hyaline membrane. There was no visible communication with the anterior chamber.

This is a true postcorneal cyst and was formed by an inclusion of Descemet's membrane with its endothelial lining. This occurred after a rupture or tear of Descemet's membrane. The gap then became filled in by a new hyaline membrane secreted from the adjacent endothelial cells.

SUMMARY

Of the fourteen cases of corneal cysts that I collected, only three were diagnosed during life. These were all of the large corneoscleral variety. In two cases, cyst formation followed an attempt to excise staphylomas; in one it occurred after a perforating ulcer; in five, after perforating injuries and in five after operative intervention. In two cases the histories were indefinite and gave no clue as to how the cysts were formed. A majority of these sections came from eyes that had been removed many years ago. It has been suggested that a possible reason for the rarity of these cyst formations in recent years is due to the fact that early enucleations are resorted to and also, that perhaps operative technic has improved.

THE ANTERIOR VITREOUS IN HEALTH AND IN DISEASE *

DANIEL B. KIRBY, M.D.
NEW YORK

A proper conception of the relations of the anterior vitreous to the crystalline lens in the human eye is of great importance for clinical and surgical understanding and guidance. As there are several mooted points on this subject, it will not be out of place at the present time to correlate the existing knowledge drawn from various sources and to bring out my observations of more than a thousand cases examined particularly for the features of this area. Since the use of the slit-lamp microscope, much discussion has arisen as to the nature of the so-called "retrolental space" and "hyaloid membrane." The behavior of features in the normal as well as in the diseased eye may be brought forward as evidence to prove that there is a condensation layer that limits the vitreous anteriorly, and that it is directly in apposition with the posterior lens capsule except for a capillary space that presumably contains a fluid like the aqueous.

To begin with, it is well to define the terms used in this discussion. The retrolental space is understood to be the dark area posterior to the human lens, as is demonstrable with the aid of the slit-lamp and microscope. It extends from the posterior lens capsule to the loose curtain that hangs down at varying distances behind the lens, which has been known by some investigators as the "front face" of the vitreous. The so-called hyaloid membrane may be defined as that extremely delicate pellicle or condensation layer that normally covers the anterior portion of the vitreous. A number of observers, in their efforts to determine the presence or absence of a true hyaloid membrane, have applied their tests to the loose curtain referred to. From my observations, the proper place to look for the so-called hyaloid membrane is directly behind the lens or at the anterior limit of the retrolental dark space. Any tests such as Koeppe [1] is widely quoted as having used should be applied to this area and not to the posterior boundary of the retrolental space.

---

* Submitted for publication, Aug. 10, 1931.

* Read at a Meeting of the American Ophthalmological Society, Asheville, N. C., June 2, 1931.

* The observations described in this article were made during the period when I held the post of Research Fellow in Ophthalmology for the American Academy of Ophthalmology and Oto-Laryngology.

1. Koeppe, L., quoted by Duke-Elder, W. S.: Brit. J. Ophth., 1930, vol. 14, suppl. 4.

EMBRYOLOGY

Mann [2] has given the most complete exposition of the origin and development of the vitreous in the human eye. The anterior vitreous includes the primary and part of the secondary vitreous. The primary vitreous has a triple origin and ends at the 13 mm. stage when the cuticula or primary lens capsule completely surrounds the developing lens. The primary vitreous present at the fifth week of intra-uterine growth is represented in the adult by the vitreous that extends from the posterior lens capsule in the so-called retrolental space, through the canal of Cloquet to the prepapillary space of Martegiani. It is formed by the ectoderm of the lens plate and lens vesicle, by the optic vesicle and by the mesoderm.

Fig. 1.—The so-called "retrolental space," extending from the posterior lens capsule to the condensation layer, which separates the primary from the secondary vitreous.

To quote further from Mann, by the sixth month, the vitreous consists of primary, secondary and tertiary vitreous. The primary forms a layer of loose mesh, containing atrophic vessels of the posterior vascular capsule of the lens, and is prolonged backward to the disk along the main trunk of the hyaloid artery. The secondary vitreous, produced by the ectodermal cells of the optic cup, is completely avascular and is still in continuity with the internal limiting membrane, the capsula perilenticularis fibrosa, with which it adheres to the lens, and the wall of Cloquet's canal, and, in addition, it is sharply separated from the tertiary vitreous, which is a zonule, by a condensation on its surface.

2. Mann, Ida: The Nature and Boundaries of the Vitreous Humor, Tr. Ophth. Soc. U. Kingdom **47**:172, 1927.

THE ANATOMY OF THE ANTERIOR ADULT VITREOUS

Fuchs [3] stated that on the anterior aspect of the vitreous is a depression (fossa patellaris) in which rests the posterior surface of the lens. By its other aspects the vitreous is applied to the inner surface of the ciliary body and retina. A fibrillar structure supporting the gel of the vitreous has been described by many authors. There are many chances for error in the interpretation of fixed and stained specimens because of the changes produced in the vitreous gel by the fixation. Mann has shown that the posterior limiting layer is identical with the internal limiting layer of the retina.

The fluid of the vitreous gel is most probably exactly similar to the aqueous, and free passage of crystalloids is possible through the anterior condensation layer of the vitreous governed by the factors of surface tension and the difference in constitution of the aqueous and vitreous. The passage of the colloid molecule is not as well understood and needs more study before discussion.

The Hyaloid Membrane.—The term hyaloid membrane is clinical. It has been known to surgeons since the early days of operations for cataract. It refers to the thin anterior condensation layer that is observed covering the bead of vitreous presenting during certain operations for cataract. This so-called membrane has certain tensile strength and resistance and contains the viscous fluid part of the vitreous. If the hyaloid membrane is not put under too much tension and if it is not pricked or otherwise injured when the bead of vitreous presents during cataract surgery, and the patient's eye is gently closed, then, other conditions being favorable, the protruding mass, even though large, usually retracts within the globe and loss of vitreous is avoided. After a period of waiting, the iris pillars may be replaced. Postoperative examination of the eye may reveal all structures in normal position, with the hyaloid in a proper plane posterior to the iris. In some cases, the vitreous will remain incarcerated in the operative scar. If, however, the hyaloid is injured and opened, there occurs the well known outpouring of vitreous mass which is so difficult to control. Any effort to stem it only results in greater loss. The proper knowledge of these conditions and the method of coping with this matter of presentation of vitreous have averted disaster in the hands of experienced surgeons in a number of cases. There is, therefore, something on the anterior surface of the vitreous which is different from the mass of the vitreous and which serves to retain the viscous gel. This is too important a conception from the surgical standpoint for one to discard, because the anterior condensation layer of the vitreous does not measure up to the qualities required in the definition of the term "membrane" or "structureless membrane."

---

3. Fuchs, E., in Duane, A.: Text-Book of Ophthalmology, Philadelphia, J. B. Lippincott Company, 1919, p. 22.

Property of the Structureless Membrane.—If a structureless glass-like membrane, such as Descemet's or the lens capsule, is examined with the aid of a specially adapted microscope using crossed Nicol prisms in the polarizer (light source) and in the analyzer (microscope) with the aid of a thin mica plate, there will be observed a play of colors that is characteristic. Koeppe [1] reported no play of colors in any of the condensation layers of the vitreous. I used Koeppe's instrument five years ago, and, first satisfying myself as to the play of colors in Descemet's membrane, found none in the vitreous. The test with the polarizing light was applied not only to the condensation layer, which is in contact with the posterior lens capsule, but to the condensation layers posterior to the retrolental space. The most anterior condensation layer (hyaloid) represents only a mesh of ultramicroscopic fibrils more dense in formation than those in the rest of the vitreous.

Location of the Anterior Condensation Layer.—Where is the anterior condensation layer of the vitreous (hyaloid) located in the normal eye? Before the use of the slit-lamp microscope, there was no question as to the location of the anterior boundaries of the vitreous. Now considerable doubt has arisen because Vogt,[4] Koeppe,[1] Butler,[5] Mann,[2] Morsman [6] and others have looked on the condensation layer separating the primary and the secondary vitreous, the "curtain" to be seen posterior to the retrolental space, as the so-called hyaloid membrane. This has proved disappointing because this layer has none of the properties of a membrane. Some slit-lamp microscopists (Morsman recently) have decided that aqueous surrounds the lens with an appreciable postlental layer. I think that if there is any such layer as a so-called hyaloid membrane, it should be looked for on the anterior surface of the primary vitreous, i.e., directly posterior to the crystalline lens capsule.

The Capillary Space Behind the Lens (Berger's Capillary Space).—This space may best be conceived of as a capillary space between the posterior lens capsule and the anterior condensation layer of the vitreous, limited in its circumference by the ligamentum hyaloidea capsulare. I have never been able to demonstrate this space in the normal eye with the aid of the slit-lamp microscope. The best evidence for its existence is the fact that there may be observed a thin layer of blood directly behind the posterior lens capsule in certain cases of trauma or after certain operations for cataract extraction in which the posterior lens capsule is left. The blood remains present and bright red (oxygenated) for an extraordinary length of time and does not behave like a hemor-

4. Vogt, A.: Atlas of the Slit-Lamp Microscopy of the Living Eye, translated by R. von der Heydt, Berlin, Julius Springer, 1921.

5. Butler, T. H.: Discussion on Microscopy of the Living Eye, Tr. Ophth. Soc. U. Kingdom **45**:657, 1925.

6. Morsman, L. W.: The Retrolental Space, Arch. Ophth. **1**:594 (May) 1929.

rhage anywhere else in the eye. After a time the colored material of the blood sinks and becomes crescentic. Whether or not the ligamentum hyaloidea capsulare is perforate, allowing communication with the aqueous of the posterior chamber, is not known. It may be that in the cases of trauma or operation in which the thin layer of blood has formed, a rent has been caused in the ligament allowing the blood to enter but affording no egress. It is my impression that it is not perforate and that the intravitreous pressure keeps the capillary space practically obliterated. Bruce, using Schoenberg's method, has never been able to demonstrate any definite line (Egger's) of attachment of the ligamentum hyaloidea capsulare, but has seen a diffuse apposition of the anterior vitreous to the posterior periphery of the lens.

Nature of the Vitreous.—Duke-Elder [7] and others have demonstrated the nature of the vitreous. It is a colloid gel permeated by a meshwork of fibrils. Ultramicroscopic examination proves the presence of colloid particles aggregated in the form of fibrils (micelles) closely interwoven in irregular patterns, through the minute meshes of which the vitreous gel can pass. The vitreous gel is not enclosed in definite spaces. It is free to move through the meshes of the fibrils.

The aqueous humor represents a solution of colloid particles in water (saline), while the vitreous gel represents a solution of water in colloid. When such a gel comes in contact with the aqueous humor, as it does in the normal eye and also in the eye on which operation is performed, it is normally separated from the humor by the difference in surface tension. This is true whether the normal anterior surface condensation layer is intact, as it is in the region of the ciliary body, or after a successful intracapsular operation, or whether the inner mass of the vitreous is exposed to the aqueous as after the operation of discission of the secondary membrane. In both conditions, the light impact line of separation of aqueous and vitreous can always be made out. There is no reason to believe that the fluid that is in solution in the vitreous gel is any different from the fluid of the aqueous humor, or that the condensation layer on the anterior surface of the vitreous exercises any selectivity in the matter of the passage of either crystalloid or colloid material in solution, either into or out from the vitreous. The material of which it is made is apparently no different from the fibrils in the rest of the vitreous.

Optical Illusions in the Vitreous.—Duke-Elder has an interesting explanation for the appearance of the living vitreous as seen with the aid of the slit-lamp microscope. The fibrils only are to be seen when

7. Duke-Elder, W. S.: The Nature of the Vitreous Body, Brit. J. Ophth., 1930, vol. 14, monog. supp. IV.

large numbers of them are arranged perpendicularly to the beam of light. Where the vitreous is more optically empty than elsewhere, as in the retrolental space, the arrangement is irregular, and the fibrils are not seen, but at the condensation layer that separates the primary and secondary vitreous, the wall of the so-called Cloquet canal, the parallel arrangement of the micelles is so marked, close and regular in pattern that an effect of "moire" gossamer-like silk curtains is produced. This does not mean that any such curtain or formation is present, but only that the fibrils (micelles) are viewed in such a way that it gives the impression of such a structure.

OBSERVATIONS MADE WITH THE AID OF THE SLIT-LAMP MICROSCOPE

There are a number of features to be observed directly posterior to the posterior lens capsule and in the retrolental space. These will serve to bring out significant points.

An Irregular Fibrillar Layer Posterior to the Lens Capsule.—This layer is to be observed by direct illumination and observation and is evidently not part of the posterior lens capsule. The fibrils and folds are part of the anterior surface condensation layer of the vitreous and are apparently in apposition with the posterior lens capsule.

The Arc Line.—Mann has shown this line to be part of the circle (1 mm. in diameter) of the attachment of the terminal branches of the hyaloid artery at the 48 mm. embryo stage. The fact that the diameter is the same in the adult as in the 48 mm. human embryo indicates that this stage of the embryo represents the final development of the hyaloid vascular system. It is interesting that Mann has shown that the hyaloid vessels never attach to the lens capsule itself, but blend with capsula perilenticularis fibrosa, which is always separated from the lens by the posterior vascular capsule or its remains. I believe that these vessels are never directly attached to the lens capsule but to the anterior condensation layer of the vitreous, which itself is attached to the lens capsule at the ligamentum hyaloidea capsulare.

Remnants of the Hyaloid Artery.—The principal remnants of the hyaloid artery, some of which may be found in all persons, are nasal to the center of the lens. They vary from filmy fibrils to definite opaque fibers or nodes. These latter have been known for years as "persistent hyaloid," as they are discoverable with the ophthalmoscope. The details of the remnants of the hyaloid are well and accurately known. Suffice it for this discussion to state that they appear to be attached to the anterior condensation layer of the vitreous and not to the posterior lens capsule itself as they are commonly said to be.

The Retrolental Space Not Optically Empty.—The retrolental space is never optically empty. With the Nernst lamp burning at greater than average intensity (Graves [8]) and with the micro-arc lamp (Vogt), one can be easily convinced that even in the so-called dark area back of the lens there are fibrils. They are few in appearance, because they (the ultramicroscopic micelles, Duke-Elder) run in all directions, and only where they are superimposed in numbers do they come within the limit of visibility with ordinary illumination.

The Retrolental Space Not Optically Inactive.—Graves [8] described the aqueous "flare" or "outstanding beam." It is the Tyndall phenomenon of a beam of light passing through colloidal solution. The aqueous is a true colloidal solution, while the vitreous is a colloidal gel (Duke-Elder). The visibility of a beam of light passing through them depends

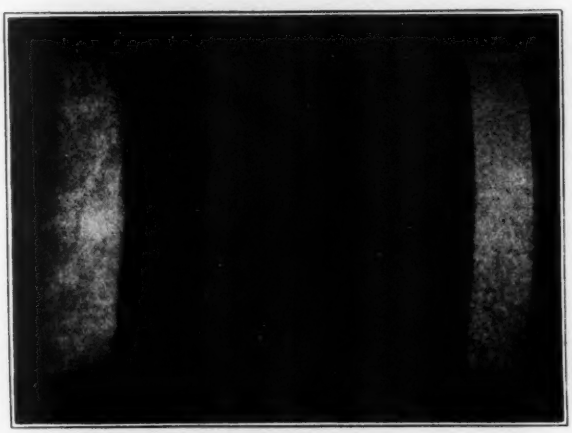


Fig. 2.—The "outstanding beam" through the normal aqueous. Nernst nitra lamp slit beam.

on the dispersion of the light by the colloidal particles. Ultramicroscopic examination demonstrates the colloidal particles, which are in constant Brownian movement in the normal state in the aqueous. The vitreous is shown to have the nature of a colloidal gel. Duke-Elder described it as first optically empty, but very soon after removal from the body it exhibits innumerable minute fibrils (ultramicroscopic micelles). These are quite free and criss-cross each other in all directions. It is the protein in colloidal solution that accounts for the relucency or visibility of the Tyndall beam through the aqueous and vitreous. It is visible in the vitreous, not only where it comes in contact with the so-called curtains or folds of the vitreous, but also in the wrongly described "optically inactive" retrolental space and in the spaces between the curtains or folds.

8. Graves, B.: The Anterior Chamber, the Aqueous Fluid and the Iris, Tr. Ophth. Soc. U. Kingdom **45**:683, 1925.

I have found no difficulty in observing the beam through the normal aqueous in the anterior chamber. Graves helped by his directions for dilating the pupil, using the 1 mm. dot beam and raising and lowering it by means of the Arruga screw of the focusing lens. Slightly eccentric vision should be used, casting the gaze to the upper or lower portion of the field of the binocular microscope while the beam is raised and lowered about the center of the field. It is not necessary to dark adapt or

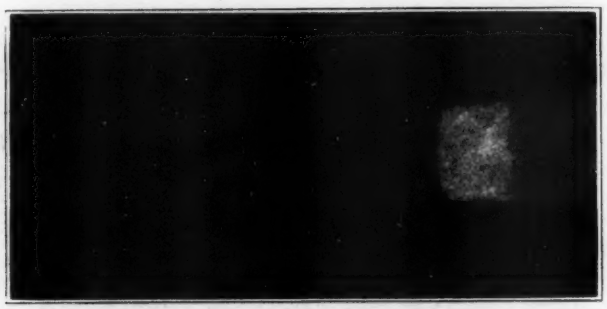


Fig. 3.—The "outstanding beam" through the normal anterior vitreous. Nernst nitra lamp 1 mm. dot beam. This beam is always more intense and more easily visible than the "outstanding beam" in the normal aqueous.

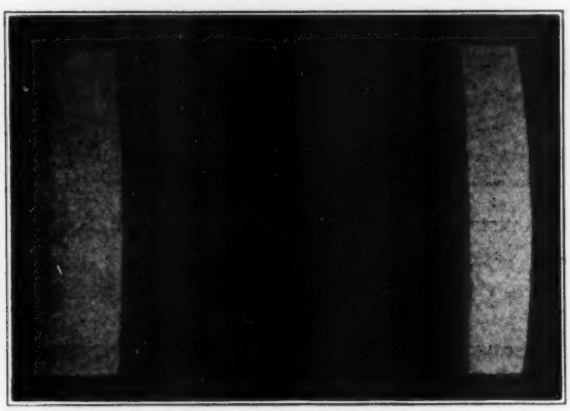


Fig. 4.—Particulate bodies floating with the convection currents in the pathologic aqueous.

to be in a perfectly dark room. With the nitra-lamp burning at slightly above average intensity, the beam is just barely visible. This is useful as a criterion for judging of any increase in its visibility in pathologic conditions.

In the retrolental space, the clear structureless portion always exhibits a greater intensity or visibility of the outstanding beam in the normal subject than does the aqueous beam of the same subject. It is always seen more easily. Roughly, the intensity of the beam is about twice as

great as that of the beam of the anterior chamber, corresponding to the relative amount of protein in colloidal solution in the two mediums. The observation that the beam is of greater intensity is all the more significant because of the fact that it is seen through the crystalline lens, which has a beam many times more intense and visible. Where the retrolental space is preserved in conditions of aphakia, the relative intensity of the outstanding beam of the aqueous and anterior vitreous can more easily be compared since there is no intervening crystalline lens, and in all instances the beam of the anterior vitreous is more intense than that of

Fig. 5.—Particulate bodies in the so-called "retrolental space" always behave as if enmeshed in a gel, such as the anterior vitreous is conceived to be.

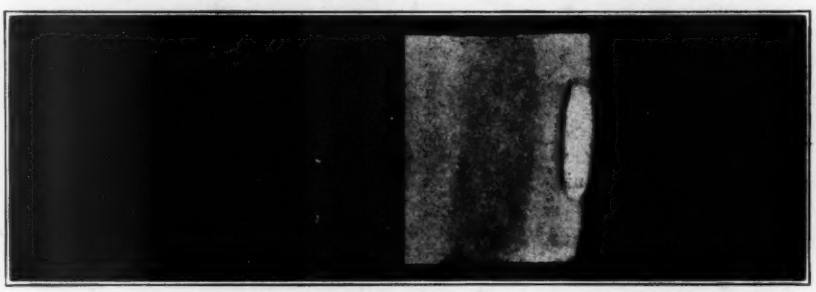


Fig. 6.—The specular reflex pattern from the posterior lens capsule region shows the influence of a minute fibrillar network, which is evidently on the posterior surface of the lens capsule. The fibrils belong to the anterior condensation layer of the vitreous, the so-called "hyaloid."

the aqueous. These facts prove that the retrolental space is not optically inactive, and that it contains a colloid that is different from that of the fluid of the anterior chamber. Therefore, aqueous such as exists in the anterior chamber is not to be found in the retrolental space.

The Specular Reflex from the Region of the Posterior Lens Capsule.—If observation is made in the axis of specular reflection from the concave mirror of the region of the posterior lens capsule, it is found that the character of the reflex varies with the position and the type and structure of the vitreous condensation layer, which is in apposition with

the posterior lens capsule. The golden reflex is not smooth and even. Its pattern is influenced, not by the lens fibers that are anterior, but by the material that is posterior to it, as is true of the mosaic of hexagonal endothelial cells on the convex posterior face of the cornea. Irregularities, folds and lines in the normal vitreous condensation layer, the arc line (Vogt has seen the arc line under these conditions as a dark fibrous stripe in luminous surroundings), the attachments and anomalies of the atrophied hyaloid artery, abnormal deposits and other features influence the specular reflex. This in itself proves that there is something in the nature of a fibrillar or irregular layer in apposition with the posterior lens capsule. It cannot be other than the anterior condensation layer of the vitreous, the so-called hyaloid membrane.

Demonstration of the Canal of Cloquet in the Adult.—Considerable credit is due Mann for her ingenious method of observing the movement of the condensation layer between the primary and the secondary vitre-

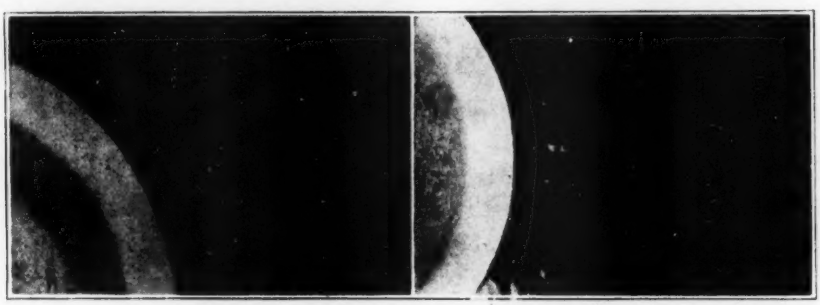


Fig. 7.—The behavior of the canal of Cloquet in the adult, as shown by the positions of its superior and inferior walls in downward and upward movements of the eye. These walls represent condensation layers separating the primary from the secondary vitreous.

ous. She selected a patient in whom this layer was easily visible, and instructed him to look down, and then back to the original position. Mann (I have confirmed every detail) found that this maneuver caused the superior wall of Cloquet's canal to be jerked violently upward, from which position it slowly settles by gravity to its original position. In a small proportion of cases an inferior wall of Cloquet's canal is brought into view. The time of the jerking upward to the full return occupies from about ten to fifteen seconds. This gave me time to investigate the horizontal boundaries of the canal. While the superior layer was still up, the patient was instructed to look to one side and then back to the primary position. This movement jerked over the side boundary so that it could be viewed. The whole procedure can be duplicated for the other side. This method of examination gives a complete idea of the boundaries of Cloquet's canal anteriorly, and it can easily be placed by infer-

ence at the region of the so-called ligamentum hyaloidea capsulare. It is not necessary to construct special microscopes to be used in the vertical position or to have the patient lie down to demonstrate Cloquet's canal in the adult. Mann's observations definitely prove that the condensation layer of the vitreous that gives the appearance of a curtain posterior to the retrolental space is really the zone of separation between the primary and the secondary vitreous.

LIQUEFACTION OF THE VITREOUS

Duke-Elder observed with the aid of the ultramicroscope the changes in the vitreous of the rabbit which occurred during the process of disintegration after the death of the animal. The gel, which was structureless at first, was soon seen to be filled with long filmy fibrils (micelles) that flitted about. Later these were seen to become granular and finally separate out in colloid dots that assumed the typical Brownian movement. In going from the gel to the solution state, the colloidal state of water in solution in protein passed over to the state of protein in solution in water.

There are innumerable grades of liquefaction of the vitreous from the normal gel state to the condition in which it has practically the consistency of water, with no viscosity. This is of practical importance at the time of operation, and knowledge of this before an operation will certainly influence surgical judgment. In cases of cataract, examination of the fellow eye, in which the opacity may be less extensive, will sometimes give valuable information.

Liquefaction of the vitreous is undoubtedly brought about through a process of enzyme digestion of the ultramicroscopic fibrillar structural framework of the vitreous. In tissue culture work, I have observed great digestive properties of fresh cells explanted from the eye. Under certain conditions that are not fully understood, this is very marked. It is the liquefaction of the fibrin of the human plasma clot that accounts for the difficulty experienced in cultivating human tissue in vitro. I think that some abnormal pathologic substances may stimulate enzyme action in the human eye and account for the lysis of the vitreous gel.

The slit-lamp microscopic changes are those of absence of a part or the whole of the typical "moire" curtains or fibrillar appearance with more or less fusion and mixing of the primary and secondary vitreous. In many cases the appearance of the retrolental space is gone, and the vitreous, which represents the mixed gel mass of the primary and secondary vitreous, is directly behind the lens. The normal attachments of the fibrils are also lost, so that the newly formed opacities float more freely and with wider excursions than do the normal.

One of the signs of disintegration of the vitreous is the appearance of fibrils that develop a higher refractive index than the normal ultra-

microscopic micelles. They are visible by property of their own structure and are not seen by massing of numerous fibrils, as are the normal vitreous fibrils. The abnormal fibers most probably represent agglutinated changed particles of the normal micelles. They are irregular in shape, knotty and arranged vertically or obliquely vertically with lesser fibrils transversely.

The vitreous dust that one sees with the ophthalmoscope most probably represents a disintegration of the ultramicroscopic micelles into the constituent granular colloid material. These aggregate into larger particles which, through a change in the colloid particles, themselves acquire a different and higher refractive index, becoming visible directly and not simply through the light that they disperse.

Floating membranes in the vitreous without fibroblastic invasion of the vitreous may occur as a result of degeneration and aggregation of fibrillar particles.

The slit-lamp microscopic appearances vary greatly. They have been sufficiently described by Vogt and others to need no repetition here. The outstanding beam of the interstitial gel remains about the same in protein content, as far as can be judged by the relucency or visibility of the beam. Duke-Elder has described the plasmoid vitreous, a condition in which the protein content of the vitreous is greatly increased.

THE BEHAVIOR OF PARTICULATE BODIES IN THE ANTERIOR VITREOUS

Particulate bodies, such as cells and cell débris, pigment granules in the retrolental space, are observed always to behave in a manner different from the particulate bodies in the aqueous of the anterior chamber. The presence of such bodies, aside from developmental remains, in the retrolental space may always be considered abnormal and indicative of a pathologic condition. Such bodies may be seen in inflammatory conditions of the uveal tract, in choroiditis, cyclitis and iridocyclitis, in hemorrhage into the vitreous, in detachment of the retina, etc. In all of these conditions, except in cases in which the vitreous has liquefied, the particulate bodies appear to be enmeshed in a gel such as the vitreous is conceived to be. Even those bodies that are very close to the posterior lens capsule behave in the same manner. With the oscillation of the globe, they are observed to gyrate in such a fashion that it is known that they are not free in such a fluid as the aqueous. Some are deposited on the inner face of the anterior condensation layer of the vitreous and as such appear to be adherent to the posterior lens capsule. I have never seen particulate bodies in the retrolental space rise and fall as they do in the aqueous. Of course, there are no convection currents in the anterior vitreous, protected as it is from the influence of the outside cold by the structures anterior to it. Nor have I seen any such bodies posterior to the lens fall as from the effect of gravity alone, as they would if they

were in an aqueous fluid. If the eye under observation is moved upward and then back to the original position, the particular bodies in the anterior vitreous will be whipped upward and then slowly fall with the fibrils of the gel in which they are enmeshed. These facts, even though they are drawn from observations on abnormal eyes, may be added to prove that the retrolental area contains a gel medium such as the vitreous is conceived to be, and that it comes directly up to the posterior lens capsule except for the capillary space previously described.

VARIOUS PATHOLOGIC CONDITIONS OF THE EYE

Iritis and Iridocyclitis.—In nearly every case of iritis, one may observe simultaneously with the appearance of particulate bodies (cells and débris) in the anterior chamber aqueous, similar bodies in the anterior vitreous. This means that nearly every case of iritis is really associated with cyclitis and that the cells in the retrolental anterior vitreous have come from the ciliary body. I have observed in a number of cases of severe iridocyclitis a condition that I have called "coagulation of the aqueous." Normally, the aqueous has extremely minute traces of fibrinogen. This amount is markedly increased in iridocyclitis owing to the inflammatory process and increased capillary permeability. A plasmoid clot may form, filling the entire anterior chamber. This clot contains numerous particulate bodies, which are observed to be motionless. Circulation of the aqueous has stopped. It is only when the inflammation has begun to clear and enzyme digestive action has cleared away or separated out the fibrin that circulation of the aqueous in its convection currents is resumed. It is difficult to dilate the pupil until the clot has been broken. A plastic fibrinous exudate is usually seen over the iris and in the pupillary area. Under such conditions one cannot observe the anterior vitreous to see whether or not any such coagulation of the anterior vitreous has occurred. If it occurs, it is different from the plasmoid vitreous that Duke-Elder described. When conditions in these cases are such that the anterior vitreous can be observed, the particulate bodies are observed as described, caught in the meshes of a gel.

In a number of cases of cyclitis, cells are seen in the retrolental space before they appear in the vitreous. Accordingly, in a case of suspected cyclitis the retrolental space should always be examined even though examination of the anterior chamber gives negative results.

Sympathetic Uveitis.—Several observers have expressed the desirability of examining the retrolental area of the uninjured eye in cases that are being watched for possible signs of sympathetic uveitis. Cells may appear here first as the initial sign of involvement. I have had no experience with such cases, but I can conceive of the information gained by the examination of this area as being most important in such suspected cases.

Choroiditis.—In certain cases of acute posterior choroiditis I have observed cells and débris forming membranes and opacities, to appear first in the posterior vitreous, later to permeate the entire vitreous, even the retrolental area, and to appear in the circulating aqueous. Such cases may occur without complicating cyclitis.

By means of phagocytic cells and enzyme action, the cells and débris may in great part be cleared from the retrolental space. It is surprising how the transparency of the vitreous may in some cases be restored by this means. It is believed that this is not a property of the anterior vitreous alone. In a number of cases in which the inflammation has apparently entirely cleared away, particulate bodies indicating remnants of cells or débris were still found in the retrolental space. The clearing is most probably accomplished by a process of lysis, digestion and phagocytosis by the tissue macrophages.

Hemorrhage into the Vitreous.—In such cases, the blood may permeate the retrolental space. Where the escape of blood has been from a retinal or choroidal blood vessel, the blood may remain confined to the posterior vitreous, or it may come forward and occupy also the retrolental area. Absorption is very slow and may leave behind for long periods minute yellowish-brown granules in the retrolental area.

Detachment of the Retina.—I have observed in every case of detachment of the retina, whether traumatic or degenerative, in which tears or holes can be seen and in a number of cases in which tears were not seen but suspected, numerous lusterful brown pigment granules in the meshes of the anterior vitreous in the retrolental area. These granules have evidently come from the area of the tear. The separation of the retina usually occurs at the pigment epithelial layer, the latter remaining attached to the choroid. The presence of these granules has helped in a number of cases to rule out tumor formation as being responsible for the detachment. In fact, if they are not present, it may be used as evidence that no tear is present. In one of my cases of contusion of the eye, two such granules were seen one month after injury. Critical ophthalmoscopic examination revealed in the periphery an area of the retina with pigment changes similar to those seen about a tear. Particular care was taken to rest the eye with atropine and a bandage to prevent a possible detachment.

Free Glioma Nodules Behind the Lens.—These peculiar whitish nodules, which have broken loose from the main mass of the glioma and live in the vitreous, obtaining their nutrition by osmosis, are often diagnostic of glioma and allied tumor formation. They may often be seen directly behind the lens by focal illumination. I have not had the opportunity to study them with the aid of the slit-lamp microscope as all of the patients were too young to submit to the observation.

Dislocation of the Lens.—One illustrative case of subluxation of the lens occurred in a man, aged 59, in whom the factor of congenital malformation was the cause for its development; there was an anterior limiting condensation layer of the vitreous in the area exposed by the shifting of the lens. The relations of the anterior vitreous were as described in normal eyes. Even after complete rupture of the attachment of all the zonular fibers to the equatorial region of the lens, it has been recognized that the lens is not completely dislocated unless the attachment of the anterior vitreous condensation layer to the posterior lens capsule by means of the ligamentum hyaloidea capsulare is also separated. This fact must be borne in mind by those who do intracapsular extractions. In favorable cases, this attachment undoubtedly separates as easily as the zonule. In fact, it is in most cases an extremely weak adhesion.

Fig. 8.—The light beam "impact" line on the so-called "hyaloid" after a successful intracapsular removal of cataract. The "retrolental space" (primary vitreous) remains intact.

VARIOUS POSTOPERATIVE CONDITIONS

After Absorption of the Lens Produced by Discission of the Anterior Capsule and Linear Extraction.—A boy of 10 years with bilateral juvenile cataracts had both lenses removed in this fashion. The relations of the retrolental space remained unchanged as the posterior capsule remained intact.

After Capsulotomy Extraction.—In these cases the same observations apply. In a number of cases observed, the relations were unaltered.

After Intracapsular Extraction.—The capsulo-zonular barrier has been held of importance to the maintenance of the normal relations of the anterior vitreous. One can readily see that this is true. After a perfect result with the intracapsular operation, the anterior face of the vitreous maintains a position definitely posterior to the iris, but in most cases there is more or less bulging forward with herniation and surging back and forth through the pupil as the eye is moved about.

After Discission of a Secondary Membrane.—It is believed from slit-lamp microscopic examinations of secondary membranes following capsulotomy extractions that it is impossible to get into the capillary space that separates the posterior lens capsule and the anterior face of the vitreous to divide the former with the secondary membrane and not injure the latter. Every type of discission of secondary membranes opens the anterior face of the vitreous, and allows the vitreous gel to herniate out into the aqueous, exhibiting a new surface layer even over the herniation.

After Incarceration of the Anterior Vitreous in Wounds.—After anterior penetrating injuries involving the anterior vitreous or after operations for cataract extraction in which vitreous has been lost, it is seldom that one does not see incarceration of the vitreous in the scar. In one case of intracapsular extraction with a loss of vitreous and a boat-shaped pupil, the anterior vitreous was drawn up to the scar, but the other relations of the anterior vitreous were undisturbed.

SUMMARY AND CONCLUSIONS

A study of the anterior vitreous of the human eye in health and disease was undertaken to correlate the existing knowledge concerning the so-called hyaloid membrane and retrolental space and to solve some of the disputed questions concerning structures and relations in this area of the human eye.

Embryologic studies by Mann demonstrate that the anterior vitreous consists of the primary vitreous, which is directly posterior to the crystalline lens in the adult, and the secondary vitreous, which is separated from the primary by a condensation layer representing the wall of the canal of Cloquet.

Surgeons are familiar with a layer known to them as the hyaloid, and anatomists have described it as being in apposition with the posterior lens capsule except for a capillary space. Doubt was thrown on this conception by slit-lamp microscopists, who found uniformly in normal eyes a retrolental dark space.

Examination of the vitreous with the aid of polarized light demonstrates that there is no structureless membrane in or surrounding the vitreous. Ultramicroscopic examination led to the hypothesis that the vitreous is a gel without any real fibrous structure. The appearance of fibrils or curtains is the result of the superimposition of numerous ultramicroscopic micelles. The structures or curtains that are seen in the normal vitreous are therefore condensation layers. The most anterior of these must represent the so-called hyaloid, and it is to be found directly back of the posterior lens capsule, separated from it only by a capillary space and kept in apposition with the posterior lens capsule by the intravitreous pressure.

Observation of the region of the posterior lens capsule, both directly and with the specular reflex, discloses a fibrillar layer immediately posterior to the capsule. The arc line and other remnants of the branches of the hyaloid artery may be found attached to this fibrillar condensation layer. They are only secondarily attached to the lens capsule through the intimate contact of the anterior condensation layer with the lens capsule at its periphery.

The visibility of the outstanding beam through the retrolental space proves that it is not optically inactive. The fact that the visibility of this beam is greater than that of the aqueous proves that it contains a colloid that is different from that of the aqueous humor. The latter as such does not exist in the retrolental space. There is no reason to believe, however, that the fluid that is in solution in the gel of the vitreous protein is of any other nature than that of the aqueous.

The retrolental dark space is proved to contain primary vitreous by demonstrating that it is the anterior portion of the canal of Cloquet. The moire curtain at the posterior wall of the retrolental space is none other than the condensation layer separating the primary and the secondary vitreous.

Liquefaction of the vitreous undoubtedly represents a lysis of the colloidal gel. This may possibly be effected by an enzyme action producing digestion of the ultramicroscopic micelles. The typical normal appearance is lost, and there is substituted a condition in which one observes a variety of formations, which represent most probably aggregations of the ultramicroscopic fibrils into dots, nodes and fibers of higher refractive index. They are observed as being suspended in a fluid of varying degrees of viscosity. Floating membranes may thus occur without fibroblastic invasion of the vitreous.

Particulate bodies in the retrolental space behave as if enmeshed in a gel such as the vitreous is conceived to be. Such bodies, except for developmental remains, may always be considered abnormal and indicative of a pathologic condition. In nearly every case of iritis, cells and débris may be found in the retrolental space simultaneous with their appearance in the anterior chamber. From this standpoint cyclitis undoubtedly is coincident with iritis. In cases of suspected cyclitis or uveitis, the retrolental space should always be examined for cells. In choroiditis, cells may come from a posterior lesion through the vitreous to the retrolental space. Clearing of the retrolental space of cells and débris is undoubtedly accomplished in the same manner as that of the rest of the vitreous by a process of lysis and digestion and phagocytosis by the tissue macrophages. In every case of detachment of the retina with tear and in those in which a tear was suspected but not seen, reddish-brown, lusterful, pigment granules exfoliated from the retinal pigment epithelium of the area of the tear have been observed in the retrolental space.

After operations on the lens in which the posterior capsulo-zonular structures are left intact, the relations in the retrolental space remain unaltered. After uncomplicated intracapsular extraction of the lens, there is to be observed a delicate condensation layer of so-called hyaloid limiting the anterior vitreous and an area that corresponds to the retrolental space. Posterior to this space, the typical moire curtain that is normally seen at the posterior border of the retrolental space may be observed in a certain number of cases. In the elderly, partial liquefaction of the vitreous is common, and normal appearances are for this reason not preserved in all cases after operations for cataract. After discission of secondary cataract, division of the anterior condensation layer is the rule, and herniation of the vitreous through the pupil is common. Even in this condition there appears an impact line of separation from the aqueous produced by the difference in surface tension of the aqueous and vitreous.

The most important conclusions are that: 1. There is a condensation layer (hyaloid) representing aggregations of ultramicroscopic micelles limiting the vitreous anteriorly. 2. This layer is in apposition with the posterior lens capsule except for a capillary space that contains a fluid presumably like the aqueous, but which is kept practically obliterated by the intravitreous pressure. 3. The retrolental space, limited anteriorly by the anterior condensation layer and posteriorly by the wall of the canal of Cloquet, contains primary vitreous.

SPONGIONEUROBLASTOMA OF THE OPTIC NERVE IN NEUROFIBROMATOSIS (RECKLINGHAUSEN) *

ISADORE GOLDSTEIN, M.D.
AND
DAVID WEXLER, M.D.
NEW YORK

Recently we reported [1] melanoma of the iris and melanosis of the uvea in a young woman who had multiple fibromas of the skin and impaired mentality. We have been fortunate since to study the optic nerves and globes in another woman who had similar generalized skin tumors of more than thirty years' duration. The orbital portion of the left nerve obtained post mortem revealed striking differences from that of the right, while the microscopic examination disclosed in this nerve a new growth, which, though undoubtedly related to glioma, presented many striking features of morphologic interest.

The literature is not lacking in reports of tumor formations in the optic nerve in neurofibromatosis, but up to the present apparently none has been considered authentic. Verhoeff,[2] while admitting that "the same influence which causes the supporting tissue of the peripheral nerve to proliferate in the form of a fibroma, may cause the supporting tissue of the optic nerve to proliferate in the form of a glioma",[1] stated that not enough evidence had been adduced microscopically to support this theory, for the two conditions should have coexisted in the same patient in at least one instance.

Earlier authors (Goldmann,[3] Byers,[4] Emanuel [5]) held that optic nerve tumors were mesodermal in origin and considered most of these tumors fibromatous in nature and probably related to neurofibroma. In spite

* Submitted for publication, Sept. 1, 1931.

* From the Laboratories and the Ophthalmological Division of the Mount Sinai Hospital.

1. Goldstein, I., and Wexler, D.: Melanosis Uveae and Melanoma of the Iris in Neurofibromatosis (Recklinghausen), Arch. Ophth. **3**:288 (March) 1930.

2. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerve, Tr. Sect. Ophth., A. M. A., 1921, p. 146.

3. Goldmann: Beitrag zu der Lehre von der Neuromen, Beitr. z. klin. Chir. **10**:13, 1893.

4. Byers: The Primary Intradural Tumors of the Optic Nerve, Studies from the Royal Victoria Hospital, Montreal, 1901.

5. Emanuel: Ueber die Beziehungen der Sehnerven Geschwülste, Arch. f. Ophth. **72**:129, 1902.

of our present knowledge that gliomas are glial in nature, and therefore ectodermal, it is significant indeed if a glioma-like tumor of the optic nerve is demonstrated beyond question in a case of generalized neurofibromatosis.

REPORT OF A CASE

History.—A woman, aged 52, who had been known to have multiple peripheral neurofibromas for more than thirty years, was admitted to the hospital on account of abdominal symptoms. Several previous operations had failed to relieve her; a diagnosis of intestinal polyposis was finally made. Death followed laparotomy.

Although careful examination of the eyes was not done, there were no external evidences of abnormality, and so far as could be ascertained there were no visual complaints. Only one fundus examination was made, and the results were reported as negative by the medical house officer.

Autopsy.—There were found skin tumors of Recklinghausen's disease, multiple intestinal adhesions and bronchopneumonia.

The skin tumors showed typical neurofibroma on microscopic examination.

Both eyes together with 10 mm. of nerve were obtained. It was noted that the left nerve was uniformly thickened in the entire course available for study; the cut section measured 6 mm. in its greatest diameter, and that of the right nerve was 4 mm. The dura was thickened to twice the width in the right nerve, while the actual width within the pia was 4 mm. compared to 3 mm. in the right. Five millimeters of each nerve was fixed separately for study in cross-section; staining was done with hematoxylin-eosin, cresyl violet, Nissl's stain, phosphotungstic acid hematoxylin, Bielschowsky's stain and Kulschitzky's stain for medullary nerve sheaths.

Microscopic Structure.—The right nerve was entirely normal in all details of its structure.

The enlargement of the left nerve within the pia was uniform in all the sections available for study. This was due to a rather sharply circumscribed tumor mass occupying about two thirds of its total area. Under low power magnification, it consisted of bundles of nerve tissue about twice the size of those in the adjacent normal nerve, separated from one another by thin poorly differentiated strands of tissue containing only an occasional elastic fiber. By comparison, the healthy nerve bundles appeared even smaller than usual, probably due to compression by the tumor. Sections stained with cresyl violet and Nissl's stains revealed most prominently, deeply staining large cells of various configurations intermingled with smaller lighter staining cells. Except for slight modifications, two types of cells were distinguished in the main:

1. A large, pear-shaped cell with prominent, fairly large vesicular nucleus, with nucleolus. Most of the cells contained clumped masses scattered irregularly through the cytoplasm. Many bore processes either short and stubby or long and fairly straight. For the most part such cells bore no process and were oval or roughly quadrilateral, but their main characteristics as to body were similar to cells with processes. Whether these were of a separate type or merely the bodies of the monopolar cells in section could not be made out. A few bipolar cells of the same character were seen. Occasionally a larger cell with two separate nuclei was found.

2. Smaller cells, with large vesicular nucleus, containing threads of reddish-staining chromatin material. Others of this group were larger and oval, with

irregular clumps in the cytoplasm, and more a triangular blunt teatlike (dwarfed) process. Some were rod-shaped. All of these cells tended to occur in small groups and had few if any processes.

The ground substance in which all the cells were embedded was a loose, fibrillar syncytium. In sections stained with phosphotungstic acid hematoxylin, parallel groups of glia fibers, containing a few cells, were seen along the poorly staining

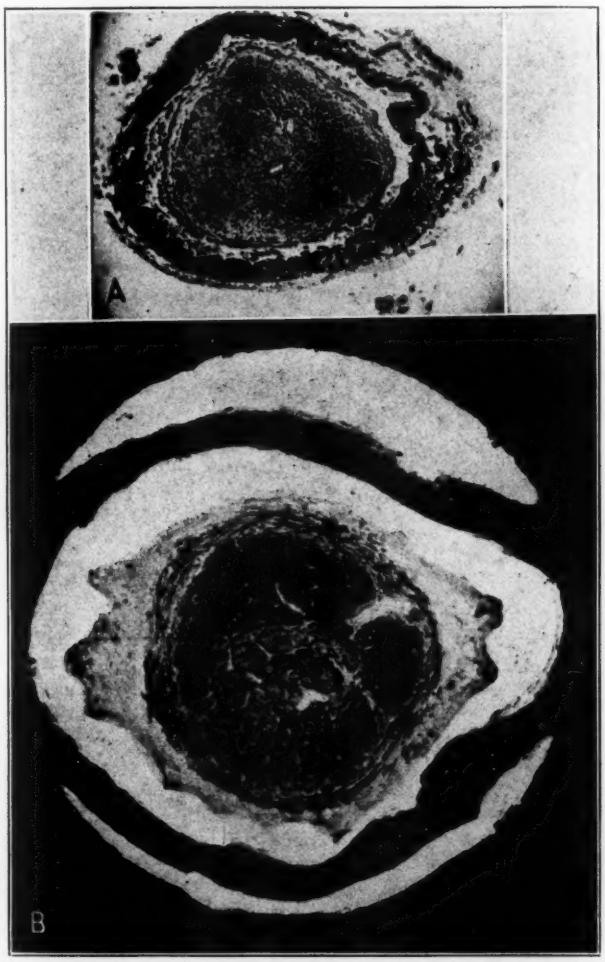


Fig. 1.—Section of the optic nerves from 4 to 5 mm. behind the globe; × 10. *A* is taken from the right eye; *B*, from the left eye. The broken line indicates the extent of the new growth.

septums. The latter stained feebly with eosin, and not at all with elastic tissue dyes. No apparent increase in the number of blood vessels over the normal was noted in the tumor. Neither vacuolization of cells nor "cytoid bodies," as described by Verhoeff,[2] were made out, and there was no tendency to hemorrhage or necrosis. The medullary sheaths stained normally in all portions of the nerve outside the tumor.

Fig. 2.—Typical section of the growth, showing numerous deeply staining large cells and neuroglia fibrils, *F*, prominent at the edge of poorly defined bundles. Phosphotungstic acid hematoxylin stain.

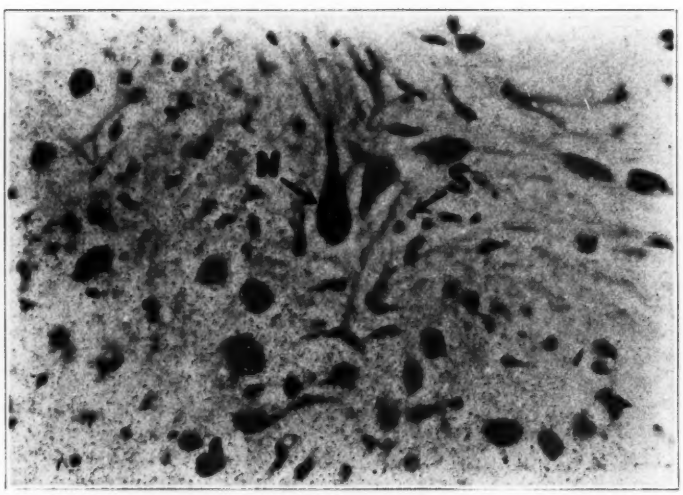


Fig. 3.—Large nerve cells (*N*, neuroblasts) and smaller round cells (*S*, spongioblasts) in oil immersion. Cresyl violet stain.

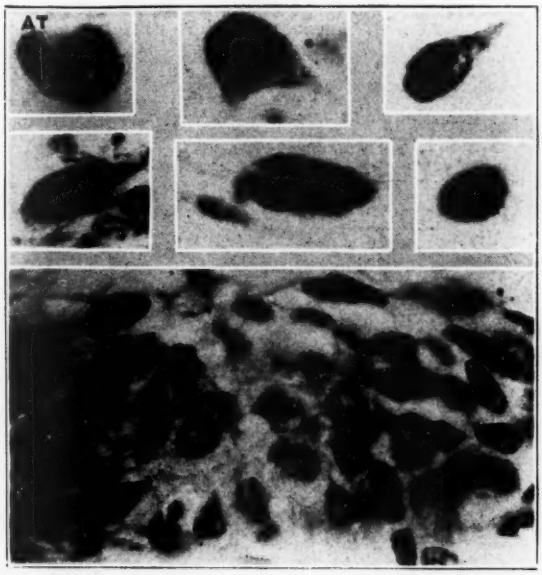


Fig. 4.—Predominant cells in the tumor: The small sections are neuroblasts; *AT*, a cell with two nuclei. The lower photomicrograph shows spongioblasts, a group of piriform cells with short processes. Cresyl violet stain.

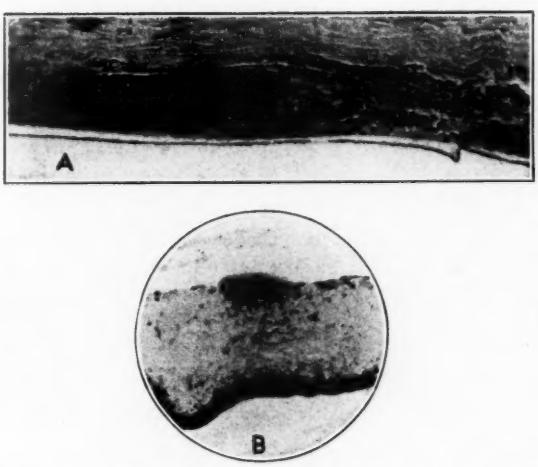


Fig. 5.—Melanosis: *A*, diffuse melanosis of the choroid; *B*, acellular melanoma (melanotic nevus) of the iris.

The pia was thickened by an increase in its normal constituents. Here and there were clusters of the small, oval type of cells assuming a nest arrangement. None of the larger cells was found in this layer. The dural space was free. The dura itself was thickened by proliferation of its tissues, but no abnormal cells were seen throughout its circumference.

The choroid was heavily pigmented throughout, especially in its outer layers. In places, as in figure 5*A*, the entire layer was so thickened with pigment as to cause a perceptible increase in its width.

The iris contained a few melanotic nodules on its anterior surface. These were confined chiefly to the pupillary zone, and were acellular. None was found near the angle of the anterior chamber as reported in our first case of neurofibromatosis.[1]

From the microscopic observations, it was evident that the cells met within this tumor were unlike those usually described for glioma of the optic nerve. There were none that could be classed as the spindle type, and while an occasional small cell could be classed as glia the large nerve cells and those of the round and piriform type appeared to require further differentiation. The cells with long processes bore a striking resemblance to ganglion cells. Sections were submitted to Dr. Joseph H. Globus of the department of neuropathology. It was his opinion that the larger cells were "neuroblasts," representing an intermediate stage in the differentiation in these nerve cells, while the smaller cells were "spongioblasts," precursors of glia cells in the derivation of these cellular elements. He suggested that the term "spongioneuroblastoma" be applied to the tumor.

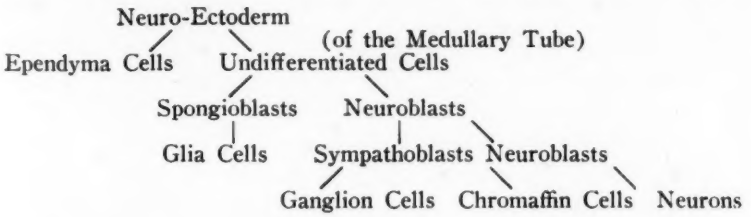
By way of explanation of this term, which utilizes the basic histogenetic principles operating in the formation of the tumor, we quote the following from Globus and Strauss:[6]

Gliomas, no matter what their histologic features may be, constitute a large and important division among the true neuromas, and therefore are not only to be considered as neuro-ectodermal in derivation, but ultimately must be traced to that division of the primitive neuro-ectoderm which is destined to develop into the glial components of the central nervous system. It is also true that this division of the primitive neuro-ectoderm, similar to the neuroblastic division, undergoes many changes before the final, fully differentiated end-product—the glia cell—is reached. It is therefore apparent that in the course of differentiation many transition stages will be encountered.

In the main, these stages of differentiation of various components of the primitive neuro-ectoderm into the mature cellular elements of the central nervous system are represented in the outline of Globus and

6. Globus, J. H., and Strauss, I.: Spongioblastoma Multiforme: A Primary Malignant Form of Brain Neoplasm, Arch. Neurol. & Psychiat. **14**:139 (Aug.) 1925.

Strauss. It will be seen that early in the histogenetic process there is a



split into two main divisions: the neuroblasts and the spongioblasts. The spongioblasts are the parent cells of the glia, while the neuroblasts develop into the various neuron forms of the cerebrospinal axis. Other neuron derivatives, such as migrating sympathoblasts and the so-called chromaffin cells, should, perhaps, be added for completion.

Following its introduction by Strauss and Globus,[7] the term "spongioblastoma" has been used to include a variety of cellular gliomas exhibiting variation in their cellular make-up and arrangement for which many specific descriptive terms had been employed. Ewing [8] described the "glioma ganglionaire," characterized by large cells resembling ganglion cells. Regarding the occurrence of such cells in optic nerve tumors, it is likely that several authors have come on them without classifying them as such. Hudson [9] spoke of cells with "oval or elongated nuclei and cytoplasm prolonged at the poles into one or two broad processes, some straight and others corkscrew-like." Martin Cohen's [10] case in a boy of 18 was reported by Dr. Ewing as a "ganglionic neuroglioma or glioblastoma, a tumor of epiblastic origin whose cells are differentiated into neuroglia cells with ganglion cells in addition." Verhoeff,[11] who examined sections of this case, disagreed with this interpretation, stating that although many of the large cells bore long processes causing a resemblance to ganglion cells, they did not contain Nissl bodies and showed numerous minute processes extending out into the surrounding tissue. Almost all showed degenerative changes such as vacuolization of the cytoplasm, an appearance typical of other gliomas of the optic nerve which he reported. He considered these cells merely large neuroglia cells. As a further evidence that tumors of the optic nerve should not be expected to contain ganglion cells, he offered the fact that the optic nerve never contains ependymal

7. Strauss, I., and Globus, J. H.: Spongioblastoma with Unusually Rapid Growth Following Decompression, Neurol. Bull. **1**:273, 1918.

8. Ewing: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919.

9. Hudson: Primary Tumors of the Optic Nerve, Royal London Ophth. Hosp. **18**:317, 1912.

10. Cohen, M.: Primary Intradural Tumor of the Orbital Portion of the Optic Nerve, Arch. Ophth. **48**:19, 1919.

11. Verhoeff (footnote 2, p. 25).

cells or remains of the embryonic neural canal, which form the basis for atypical gliomas of the brain containing rosettes and in some instances ganglion cells. While the latter point is well taken, it does not definitely preclude the possibility of growth from undifferentiated cells in the nerve. Further, the large nerve cells in the present case need not be taken to be mature ganglion cells, although many contain Nissl substance in their cytoplasm. In the transition from neuroblasts to the fully developed ganglion cell, and especially in the same growth, the resemblance of intermediate forms to one another is close. The presence of cells with two nuclei is in itself significant, being a feature not attributed to gliomas of the optic nerve. Another feature, the tendency to the clustering of cells, seen in the tumor, as well as in the pia, is typical of both neuroblastomas and spongioblastomas (Globus and Strauss [6]).

COMMENT

Clinically, the case reported is unique among tumors of the optic nerve in that attention to it was not called by the usual symptoms—those of defective vision and exophthalmos. That it did not produce such symptoms was due partly, of course, to the confinement of the growth to the nerve. In discrediting the theory that glioma of the optic nerve may be neurofibromatous in nature, Hudson [9] pointed to the early loss of function produced by atrophy of nerve fibers in optic nerve tumors as opposed to the minimal disturbance of function characteristic of peripheral neurofibromas. According to Hudson's conception, the lack of destruction of the nerve in our case would be positive evidence of similarity between the growth in the nerve and of other neurofibromas. This, then, would be another reason for the benign course of the optic nerve lesion.

While fundus lesions characteristic of, or even suggestive of, neurofibroma, have not been demonstrated in neurofibromatosis, it is interesting to note that such new growths have been found in an allied disease, tuberous sclerosis. This condition, marked by sclerosis or hardening of portions of the cortex of the brain, hard solid tumors projecting into the ventricles and cyst formations, has been regarded by Bielschowsky as a form of diffuse spongioblastosis. The close association of the disease with Recklinghausen's has been insisted on by several authors (Orzechewski and Nowicki, Bielschowsky) who proposed an inclusive term as "gliomatosis or spongioblastomosis centralis, peripheralis or universalis," depending on the localization of the nerve structures involved. Moreover, it appears that the tendency to simultaneous growths in other parts of the body, such as lipoma, fibroma and sarcoma, is only slightly less marked in neurofibromatosis than in tuberous sclerosis.

As regards the ocular lesions in tuberous sclerosis, van der Hoeve [12] in 1921 described typical lesions in the fundus in several cases of tuberous sclerosis. These consisted of well marked knoblike tumors of the disk and flat swellings in the retina. Histologically, they consisted chiefly of cells probably derived from the primary retinal anlage, a sort of neurocyte that had not differentiated into ganglia or glia cells. The relation of the disease to Recklinghausen's was brought home to van der Hoeve [13] through his discovery of similar lesions in the fundus of a patient who had tumors of the acoustic nerve and neurofibromas of the back. This patient exhibited, among other lesions in the fundi, two definite swellings in either eye which resembled those he had seen in tuberous sclerosis. It remains only for microscopic evidence in this regard to substantiate the connection between the diseases as far as the retina is concerned.

12. van der Hoeve: Augengeschwülste bei der tuberösen Hirnsklerosen (Bourneville) und verwandten Krankheiten, Arch. f. Ophth. **105**:880, 1921.

13. van der Hoeve: Augengeschwülste bei der tuberösen Hirnsklerose, Arch. f. Ophth. **111**:1, 1923.

OCULAR SYPHILIS

II. FACTORS INFLUENCING THE LOCALIZATION OF SYPHILIS IN THE EYE *

JOSEPH V. KLAUDER, M.D.
Dermatologist and Syphilologist to Wills Hospital
PHILADELPHIA

My purpose in this paper is to review laboratory and clinical data relating to circumstances concerned in the localization of syphilis in the eye.

Factors influencing the localization of any infection, syphilis being no exception, concern strains of organism; tissue predisposition; hereditary and racial influences; conditions that establish a locus minoris resistentia, such as trauma; immunologic response of the host and circumstances that alter this response, such as toxic states, presence of other infections and the allergic state; psychic-mental stress and strain.

STRAINS OF SPIROCHAETA PALLIDA

The possibilities of the existence of strains of Spirochaeta pallida were first suggested in 1912 by Noguchi [1] and later supported by the experimental study of Nichols [2] and by Reasoner.[3] Nichols isolated from the spinal fluid of a patient with early neurosyphilis, a strain of organism which usually produced ocular lesions in rabbits. In 1919, Levaditi and Marie [4] isolated strains of organisms that they regarded as a dermotrophic strain and a neurotrophic strain.

Subsequent studies of syphilis in the rabbit have failed to demonstrate the existence of diverse strains of Spirochaeta pallida endowed with the capacity of becoming localized in different organs.[5]

---

* Submitted for publication, Sept. 3, 1931.

* From the Wills Hospital Clinic for the Treatment of Ocular Syphilis and the Research Institute of Cutaneous Medicine.

1. Noguchi, H.: Morphological and Pathogenic Variations in Treponema Pallidum, J. Exper. Med. **15**:201, 1912.

2. Nichols, H. J.: Observations on a Strain of Spirocheta Pallida Isolated from the Nervous System, J. Exper. Med. **19**:362, 1914.

3. Reasoner, M. A.: Some Phases of Experimental Syphilis, J. A. M. A. **67**: 1799 (Dec. 16) 1916.

4. Levaditi, C., and Marie, A.: Étude sur le tréponéme de la paralysie générale, Ann. Inst. Pasteur **33**:741, 1919. Marie, A.; Levaditi, C., and Banu, G.: Transmission expérimentale du tréponéme de la paralysie générale (virus neurotrope) par contact sexuel, Compt.rend. Acad. d. sc. **170**:1021, 1920.

5. For a more detailed discussion see Truffi, M.: Immunity, Superinfection, Reinfection in Syphilis, Tr. Eighth Internat. Cong. Dermat. & Syphil., Copenhagen, 1930, p. 194; ibid. Ann. de dermat. et syph. **1**:689 (July) 1930.

Relative to the existence of strains of Spirochaeta pallida, Brown[6] stated:

> The behavior of a given strain is by no means constant. Spirochetes vary in their action just as animals do. The rate of passage of stock strains, the state of the infection at the time of passage, the source of material and the site of inoculation are all potent factors in determining the ultimate effects which the organisms produce.

It therefore appears that there is little experimental status to the existence of an ocular strain of Spirochaeta pallida.

From a clinical visualization of syphilis in general, the existence of different strains of Spirochaeta pallida is not suggested. Instances of conjugal dementia paralytica and tabes are rare as is likewise the frequently quoted series of cases by Mattauschek and Pilcz, in which five Austrian Army officers, infected by the same woman, developed dementia paralytica.

Clinical manifestations of syphilis vary considerably in patients whose infection is contracted from the same source. One rarely sees ocular lesions of syphilis in the spouse of a patient with such localization. This occurred, however, as reported by Escher,[7] in three members of the same family; a man, his wife and his mother-in-law developed iritis in the secondary stage of syphilis. In 100 patients with interstitial keratitis at the Wills Hospital Clinic, the disease was present in brothers or sisters in only five instances.

ATTEMPT TO ISOLATE AN OCULAR STRAIN OF SPIROCHAETA PALLIDA

The following study was undertaken to obtain an ocular strain of Spirochaeta pallida. The opportunity of obtaining viable Spirochaeta pallida from patients with ocular lesions of syphilis sufficient for rabbit inoculation is rare, hence this report of one case is justified:

> A man, aged 32, attended the Wills Hospital Clinic for treatment for acute iritis which was regarded as syphilitic in origin. He presented a chancre on the left arm (fig. 1), the dark-field examination of which disclosed many motile spirochetes, a sparing macular eruption of secondary syphilis, and a positive Wassermann reaction of the blood (4 plus). The chancre was excised in toto, ground in sterile saline solution and immediately injected into the testes of three rabbits. After an incubation period of forty-five days, the animals developed typical syphilomas of the testes which contained motile Spirochaeta pallida. Ten days after the appearance of the syphilomas, bilateral castration was performed, and another series of animals were inoculated. In this way the infection was transmitted to four generations of rabbits. In some animals, castration was performed forty-eight hours after inoculation; in others, twenty days after inocula-

6. Brown, W. H.: Syphilis, Immunity, Reinfection, Superinfection, Tr. Eighth Internat. Cong. Dermat. & Syphil., Copenhagen, 1930, p. 220.

7. Escher: Trois cas de syphilis dans une même famille, Ann. de dermat. et syph. **2**:454, 1921.

tion. Unilateral, as well as bilateral castration, was performed. Although all the animals developed testicular lesions, in none did the infection become generalized nor did ocular lesions develop.

IMMUNOLOGIC RESPONSE

The extensive studies of Brown and Pearce [8] furnish evidence that immunologic response of the rabbit to syphilitic infection is a factor in determining ocular localization in this animal.

Preliminary to discussing their experiments, it is desirable to state that the mechanism of immunity in syphilis is apparently entirely cellular,[9] and the most successful way of infecting rabbits with syphilis is by means of testicular inoculation. If the testicular syphiloma is undisturbed, rarely does the infection become generalized. The local

Fig. 1.—Chancre on the arm of a patient who had an eruption of syphilis and iritis. The chancre was excised, emulsified and injected into testes of rabbits in the direction of isolating an ocular strain of Spirochaeta pallida as detailed in text.

area of infection constitutes, it is thought, an immunologic depot which inhibits the dissemination of the infection.

Brown and Pearce [8] showed that interference with the development of the primary lesions in rabbits, by early or late removal and by subcurative injections of the arsenicals, favored the outbreak of generalized lesions. They demonstrated [10] that generalization of the infection

8. Brown, W. H., and Pearce, L.: Influence of Light on Reaction to Infection in Experimental Syphilis, J. Exper. Med. **45**:497, 1927; Experimental Production of Clinical Types of Syphilis in the Rabbit, Arch. Dermat. & Syph. **3**:254 (March) 1921.

9. Klauder, J. V.: The Humoral Aspects of Immunity to Syphilis, Arch. Dermat. & Syph. **23**:884 (May) 1931.

10. Brown, W. H., and Pearce, L.: Animal Resistance and the Endocrine System of the Rabbit in Experimental Syphilis, Proc. Soc. Biol. & Med. **20**:476, 1923.

tended to follow a certain order, that is, bone lesions, if they occurred, were likely to follow lesions of the testes or scrotum, while keratitis and iritis occurred toward the end of the cycle of tissue involvement, the skin and mucous membranes occupying an intermediate position. By early and radical interference it was possible to so alter the course of the disease as to cause bone lesions practically to supplant those of the testes in the defensive reaction; conversely, by permitting the testicular reaction to progress to a given point, it was possible to confer protection on this group of structures. In this event no bone lesions occurred but the generalized disease began with the involvement of the next group of tissues in order, producing an infection that was essentially one of the skin, mucous membranes and eyes.

Brown and Pearce thus demonstrated that the occurrence or nonoccurrence of eye lesions was determined by the prior course of the infection. If an animal had severe secondary bone and skin lesions, eye lesions in such animal were rare, i. e., the immunity was such as to protect the eye from developing lesions. On the other hand, if there were very few bone or skin lesions, eye lesions were constant.

It was observed that, although inoculation into both testes seemed to confer protection, which extends in most cases to other tissues, it may not reach the eye. The eye and the nervous system were afforded less protection by tissue reaction taking place elsewhere.

CLINICAL CORRELATION OF EXPERIMENTAL OBSERVATION

From the study of Brown and Pearce, it appears that the nature of the tissue reaction to syphilis in the rabbit is a factor in determining ocular localization.

It is desirable to correlate these observations with clinical data regarding ocular syphilis, with particular reference to the involvement of the nervous system.

In the foregoing regard it is interesting to note that many years ago that astute clinical observer, Fournier,[11] regarded iritis in secondary syphilis as an unfavorable prognostic sign, so far as the subsequent course of the infection was concerned.

Of 40 patients with syphilitic iritis whose ultimate course was observed by Trousseau,[12] 23 later developed frank neurosyphilis and 2 others probably died of cerebral syphilis. In a similar study by Igersheimer [13] of 18 patients, 2 later developed neurosyphilis and 3 probably had such involvement. On the other hand, of 79 patients with syphilitic iridocyclitis and 42 patients with choroiditis, reported by

11. Fournier, A.: Traité de la syphilis, Paris, Rueff et Cie, 1899.

12. Trousseau, M. A.: Valeur prognostique de l'iritis dans la syphilis, Ann. d'ocul. **123**:358, 1900.

13. Igersheimer, J.: Syphilis und Auge, Berlin, J. Springer, 1918, p. 294.

Snydäcker,[14] none subsequently developed tabes or dementia paralytica. Mattauschek and Pilcz,[15] from their studies, concluded that a uveitis is of no prognostic significance in regard to the subsequent development of neurosyphilis. Of 73 patients, who at one time in the course of their infection had uveal disease, 8, or 10.9 per cent, developed frank neurosyphilis.

Wile and Marshall[16] observed a high percentage of spinal fluid abnormalities in patients with iritis in untreated secondary syphilis. In their series of 508 patients with secondary syphilis, 21 had iritis, 15 of whom, or 71.4 per cent, had abnormal spinal fluids. Of the entire 508 patients, 35, or 6.9 per cent, had abnormal spinal fluids.

On the other hand, Zimmermann[17] concluded from his study that the incidence of asymptomatic neurosyphilis does not appear to be higher in patients with iridocyclitis than in syphilitic persons in general. In 61 patients with early and late iritis, partially treated, 9, or 15 per cent, presented spinal fluid abnormalities. He compared this percentage with Moore's[18] study of the spinal fluid in treated syphilis. Among 377 white patients in all stages of the disease, 60, or 15.9 per cent, had abnormal spinal fluids; of 265 Negroes, 22, or 8.3 per cent, showed abnormalities.

In Moore's study, 20 per cent of 111 patients with early iritis and 26 per cent of 109 patients with late iritis had positive Wassermann tests of the spinal fluid. This percentage is not greater than that reported by others in syphilitic patients without iritis.[19]

A dictum propounded by Fournier to the effect that patients with pronounced secondary eruptions were less likely to develop neurosyphilis later is in line with Brown and Pearce's observations, as well

---

14. Snydacker, E. F.: Absence of Iritis and Choroiditis Among Syphilitics Who Have Become Tabetic, J. A. M. A. **54**:933 (March 19) 1910.

15. Mattauschek, E., and Pilcz, A.: Beitrag zur Lues- Paralyse-Frage, Ztschr. f. d. ges. Neurol. u. Psychiat. **8**:133, 1911.

16. Wile, U. J., and Marshall, C. H.: A Study of the Spinal Fluid in One Thousand Eight Hundred and Sixty-Nine Cases of Syphilis in All Stages, Arch. Dermat. & Syph. **3**:272 (March) 1921.

17. Zimmermann, E. L.: Syphilitic Iridocyclitis with a Consideration of Factors Influencing Its Occurrence, Arch. Ophth. **53**:549 (Nov.) 1924.

18. Moore, J. E.: The Cerebrospinal Fluid in Treated Syphilis, J. A. M. A. **76**:769 (March 19) 1921.

19. A number of investigators have reported varying incidences of papillitis in patients with positive spinal fluids in the early stage of syphilis. Since papillitis can be regarded as an expression of neurosyphilis, a discussion of it is not the purpose of this paper. Memmesheimer and Lunecke's report (Liquorpositive Luesfälle im Frühstadium und ihre Augenveränderungen, Dermat. Ztschr. **39**:213, 1923) is interesting in this regard. In a detailed study of 50 patients with positive spinal fluids in early syphilis, 33 presented ocular alterations, with particular reference to the presence of papillitis, and alteration in the papillary reflex, as determined by the Hess pupilloscope and the presence of Haenel's symptom.

as with other immunologic studies, which emphasizes the rôle of the skin as a defensive organ.

To correlate the foregoing statement clinically, one would expect a high incidence of iritis with sparing eruptions of syphilis. In Moore's [20] study of 111 patients, whose iritis was associated with a secondary eruption, he observed about an equal number of patients with severe or intense secondary lesions as compared to those with trivial or insignificant lesions. He concluded that the proportion of mild, moderately severe and severe secondary eruptions seems about the same as in a series of patients with secondary syphilis without iritis.

Zimmermann [21] and also Moore [20] observed that a folliculopapular syphilid was the most frequent type of secondary eruption associated with iritis in the Negro. Although this variety of syphilid is characteristic of the Negro, and uncommon in the white person, it is much more frequent in association with iritis than without it. Both investigators pointed out that a common clinical syndrome in the Negro comprises a profuse folliculopapular eruption, definite syphilitic arthritis (usually of the ankles), marked polyadenitis and iridocyclitis. No other important conclusions could be drawn by Moore from his clinical study as to race or sex or as to the association of other lesions with early syphilitic iritis.

The studies of Brown and Pearce are particularly interesting in relation to interstitial keratitis. Guy [22] very appropriately pointed out the following parallel clinical facts regarding interstitial keratitis: Syphilis in females is usually less malignant than in males. Interstitial keratitis is more common in females than in males. Persons with congenital syphilis who survive do so by virtue of the development of immunity to a widely disseminated infection. Syphilitic interstitial keratitis usually occurs as a manifestation of congenital syphilis.

The well known fact that syphilis is less malignant in the female, hence causes less tissue reaction and affords less protection to ocular structures, is a more reasonable explanation than other given theories, I believe, of the greater incidence of interstitial keratitis in females.

It is a common teaching and a general belief that active tertiary lesions are invariably confined to one organ; it is uncommon to see active lesions at the same time in two different systems or organs. For example, it is believed that patients with frank tabes or dementia paralytica are rarely seen with active tertiary lesions in the splanchnic

20. Moore, J. E.: Syphilitic Iritis, Am. J. Ophth. **14**:110, 1931.

21. Zimmermann, E. L.: Comparative Study of Syphilis in Whites and Negroes, Arch. Dermat. & Syph. **4**:75 (July) 1921.

22. Guy, W. H.: Interstitial Keratitis in Congenital Syphilis, J. A. M. A. **87**: 1551 (Nov. 6) 1926.

system or in the skin and bones. This is held by some as evidence of the existence of strains of spirochetes. The more reasonable explanation, however, is that this clinical evolution of syphilis corroborates experimental observation that local tissue reaction plays an important rôle in the defensive mechanism of syphilis, acting, so to speak, as an immunologic depot, holding in check progress of the disease in other organs, and that clinically, the disease expends itself at any given time in one organ but may later become active in other organs, the infection pursuing, as in experimental rabbit syphilis, a cyclic course.

The studies of some investigators of late ocular syphilis are consistent with the belief that late active syphilis is usually confined to one organ. For example, in Zimmermann's [17] series of 82 patients with late iritis, only 3 presented frank tertiary lesions in other organs. Igersheimer [18] stated that late iritis is usually seen as an isolated manifestation of the disease. Among 156 patients with iritis, studied by von Schroeder,[23] only 2 presented tertiary lesions in other organs, and in Widder's [24] study of 46 patients with iritis, secondary lesions were found in 37 and tertiary lesions in only 1.

Interstitial keratitis is invariably the only manifestation of active syphilis. In 100 patients with this disease studied at Wills Hospital Clinic, no other active syphilitic process was demonstrable.[25]

Within recent years, since the syphilitic patient has been so intensely studied clinically and the spinal fluid so frequently examined, a number of reported studies have shown the association of syphilitic processes in different organs to a much greater extent than formerly taught. For example, in Wile and Marshall's [16] study it was observed that in 155 cases of late syphilis, involving the skin and mucous membranes, the bones and the viscera, in the form of gummas and diffuse syphilomas, 48 patients, or 30 per cent, showed involvement of the nervous system at the same time. In Moore's [20] study of syphilitic iritis, 50 per cent of the patients with late iritis had manifest lesions of late syphilis.

Figure 2 is the photograph of a patient who had an iridocyclitis and at the same time a nodular syphilid on the forearm. He also had an inactive neurosyphilis—neurologic abnormalities, the absence of subjective symptoms of neurosyphilis, with a normal spinal fluid.

---

23. von Schroeder, T.: Beitrag zur Kenntnis der Iritis syphilitica, Diss., Dorpat, 1880.

24. Widder, J.: Ueber Iritis Syphilitica, Arch. f. Ophth. **27**:99, 1881.

25. The stigmas of congenital syphilis presented by these patients cannot be regarded as due to an actively existing syphilitic process. Although the spinal fluid of not all of the patients was examined, none of that which was examined presented clinical evidence of active neurosyphilis.

THE RÔLE OF THERAPY; NEURORECURRENCE; IRIDORECURRENCE; JARISCH-HERXHEIMER REACTION

The occurrence of neurorecidive—symptoms of neurosyphilis appearing as a relapsing manifestation following insufficient treatment for early syphilis, more particularly with the arsenicals—is comparable to the condition caused by the administration of subcurative doses of the arsenicals in syphilis in rabbits as a means of generalizing the infection. In both instances the mechanism is probably the same, namely, insufficient treatment interferes with the immunologic response of the host to the infection. Indeed, it has been stated that insufficient treatment in early syphilis, followed by a lapse of treatment, may produce more disastrous results than if no treatment were given.

Fig. 2.—A nodular syphilid. The patient also had iridocyclitis and inactive neurosyphilis—neurologic abnormalities, absence of subjective symptoms of neurosyphilis, with a normal spinal fluid. The association of ocular lesions of syphilis with syphilitic lesions in other organs is discussed in the text.

Since Brown and Pearce showed that the eye and the nervous system are afforded less protection by tissue reaction taking place elsewhere, it is proper to place ocular lesions of syphilis, iridorecurrence, occurring after insufficient treatment in early syphilis, in the same category as neurorecurrences.

Neurorecurrence is manifested as an acute syphilitic meningitis. The most frequent concomitant ocular lesions are optic neuritis, papillitis, involvement of the oculomotor, abducens or trochlear nerves. Zimmermann [17] reviewed the literature of iridorecurrences. A detailed review here is not necessary. He quoted isolated reports of iridorecurrences in which different ocular structures were involved, occurring with or without syphilitic meningitis.

Significant, in the foregoing visualization of iridorecurrence, is Moore's [20] finding of 41 per cent positive Wassermann tests in the

spinal fluid of patients with relapsing iritis. In 4 of 17 patients with relapsing iritis, there was associated outspoken neurorecurrence, and in 3 others, the iritis was immediately preceded or followed by a neurorecurrence. Zimmermann noted this same association in 2 of 15 cases observed by him. As Moore stated, when these figures are combined with Zimmermann's figures, this gives an incidence of neurorecurrence associated with relapsing iritis, of 20 per cent, whereas the normal expectancy of neurorecurrence, as a complication of relapsing mucocutaneous secondary syphilis, is only 5 per cent (Moore and Kemp [26]).

Iridorecurrence was formerly more frequent in that era of arsphenamine therapy when it was thought that relatively few injections of the arsenicals constituted sufficient therapy. Since harmful influence of insufficient treatment for early syphilis is now generally recognized, the incidence of iridorecurrence has become considerably less.

Soon after the introduction of the arsenicals in the treatment for syphilis, aggravation of preexisting syphilitic lesions was observed immediately following the administration of large doses of the drug. This phenomenon is known as the Jarisch-Herxheimer reaction [27] and deserves brief discussion since this reaction is given to explain the appearance of syphilitic ocular lesions following the use of the arsenicals. The appearance of such lesions in areas that were apparently uninvolved is generally believed to be due to the activation of dormant Spirochaeta pallida.

Zimmermann [28] reviewed the literature regarding the occurrence of the Jarisch-Herxheimer reaction in relation to ocular lesions of syphilis and quoted a number of reports in which preexisting lesions of syphilis were aggravated or new lesions appeared. These reports embrace ocular lesions of divers nature.

The Jarisch-Herxheimer reaction, so far as it concerns ocular lesions of syphilis, is seen considerably less frequently than formerly, since the possible harmful effects of initial injections of large doses of the arsphenamines is now generally known. For example, the reaction has never been observed among the number of patients receiving antisyphilitic

26. Moore, J. E., and Kemp, J. E.: The Treatment of Early Syphilis; II. Clinical Results in 402 Patients, Bull. Johns Hopkins Hosp. **39**:16, 1926.

27. The reaction as originally described was an accentuation, reddening and swelling of macular and papular rashes, occurring within twenty-four hours after the use of mercury. Its occurrence, which is more frequent after the use of the arsphenamines, was given as a further proof that these drugs exert a powerful direct effect on the spirochete. Before the discovery of Spirochaeta pallida, the reaction was frequently used for diagnostic purposes (a diagnosis by the method of ex nocentibus) in order to determine whether isolated skin lesions that were only just visible might be pronounced roseola or not. For a more detailed discussion of the Jarisch-Herxheimer reaction, see Klauder, J. V.: Early Neurosyphilis Asymptomatica; With Report of Observations and Cases, Am. J. Syph. **3**:559, 1919.

28. Zimmermann, E. L.: The Rôle of the Arsphenamines in the Production of Ocular Lesions, Arch. Ophth. **57**:509 (Sept.) 1928.

treatment in the Wills Hospital Clinic. In acute cases, initial treatment consists of either intramuscular injections of bismuth or small doses of the arsphenamines.

TISSUE PREDISPOSITION

Of ocular structures, the cornea [29] both in congenital syphilis and in experimental syphilis in rabbits is conspicuous in relation to tissue predisposition of Spirochaeta pallida.

The cornea shares with lymphatic tissue, testes and bone marrow as having the greatest tissue predisposition for Spirochaeta pallida. There is no experimental or clinical data that furnishes a clue in the explanation of the absence of tissue predisposition of the cornea in acquired syphilis. It has been pointed out by some writers that the cornea of children, in whom syphilis is acquired subsequent to birth, is still prone to interstitial keratitis.

Enroth [30] presented evidence tending to show that the pathogenesis of stigmas of congenital syphilis is intimately associated with disturbance of the endocrine glands. He expressed the belief that the susceptibility of the cornea in congenital syphilis to interstitial keratitis is in etiologic relation to endocrine gland disturbance.

In regard to the foregoing theory, Brown and Pearce [8] showed that the endocrine glands and lymphoid tissue are factors concerned in the immunologic response of the rabbit to syphilis.

Constitutional predisposition of the iris may be concerned in the already quoted observations of Zimmermann [21] and Moore [20] regarding the frequency of iritis in the Negro. Zimmermann referred to the reports of Bull, Grima and Atkinson as further evidence of the high incidence of iritis in the American Negro.

Tissue predisposition and the question of strains of Spirochaeta pallida are intimately concerned, since it cannot be defined where one begins and the other ends. The problems of tissue resistance, predisposition and elective localization of Spirochaeta pallida are complex and altogether incompletely understood.

OCULAR LESIONS IN CONGENITAL SYPHILIS

Green [31] reviewed the literature on the eye in congenital syphilis and reported his study of 100 patients with congenital syphilis. The inci-

29. It may be mentioned at this time that intracutaneous tests with an emulsion of normal human cornea were performed on patients with interstitial keratitis in order to determine if such patients were sensitized to corneal tissue. This study was in the direction of determining if allergy was concerned in the involvement of the second eye. All such tests were negative.

30. Enroth, E.: Parenchymatose Keratitis und Konstitution, Klin. Monatsbl. f. Augenh. **65**:266, 1920.

31. Green, John, Jr.: The Eye in Hereditary Syphilis, Am. J. Dis. Child. **20**: 29 (July) 1920.

dence of eye lesions in congenital syphilis has been given by Roth as 39 per cent in 127 cases, and by Fournier as 48 per cent in 212 cases, presumably including all ages of children. Igersheimer found pathologic lesions in 80 per cent of 27 nurslings. Lesions of the lids, conjunctiva, lacrimal apparatus and lens are either rare or secondary to other lesions and are relatively insignificant. Of 2,000 cases of congenital syphilis, keratitis occurred in 50 per cent. A frequent association of retinochoroiditis with keratitis has been observed, usually occurring after the keratitis. A vertically oval cornea has been claimed to be more frequent in hereditary syphilis and to be a precursor of keratitis. Iritis is infrequent as compared to acquired syphilis. Its incidence is really unknown since it is not looked for as a routine measure. A vitreous haze in childhood denotes congenital syphilis. It is often associated with retinochoroiditis. Optic neuritis was found by Heine in 82 per cent of 105 babies. Other observers have failed to find such a high incidence.

In Green's study of 100 syphilitic infants and children, the following ocular findings are reported: 74 showed definite pathologic conditions in one or more of the ocular tissues, or some derangement of ocular function; 9 had fixed or unequal pupils; 4 had strabismus; 1 had ptosis; 3 had nystagmus, 19 had had keratitis or evidence of past keratitis; 2 had fine haze in the vitreous, and 52 had undoubted pathologic changes in the eyegrounds. There was scarcely a single pathologic eyeground that did not present lesions of diverse types; in other words, mixed forms predominated. Of those with pathologic eyegrounds, 8 had optic neuritis; 8 had optic atrophy and 28 had peripheral and central punctate pigmentation. This pigmentation consisted of tiny dots of brown or brown-black pigment strewn fairly evenly over the retina. Between the dots are minute yellowish-red spots. Three had peripheral punctate pigmentation; 5 showed large irregular areas of retinochoroidal atrophy; 4 had masses of gray pigment alternating with depigmented areas; 2 had disseminated retinochoroiditis; 1 had punctate neuroretinitis; 2 had retinal hemorrhages, and 3 had perivasculitis. Green is convinced that the dark brown punctate pigmentation is characteristic of congenital syphilis. He found this appearance in 2 adults only, and 1 of these had scars of an old keratitis.

TRAUMA

In a clinical visualization there is much evidence that trauma plays a rôle in determining the localization of a syphilitic process.[32] The surgeon is cognizant of syphilis as a factor in the nonhealing of wounds, nonunion of fractures and the appearance of cutaneous syphilids at the

32. Klauder, J. V.: Syphilis and Trauma, J. A. M. A. **78**:1029 (April 8) 1922. Klauder, J. V., and Solomon, H. C.: Trauma and Dementia Paralytica, J. A. M. A. **96**:1 (Jan. 3) 1931.

site of surgical incisions in syphilitic patients. The laryngologist is reluctant to perform a septum operation on a person with syphilis. Trauma of the head can be ascribed as a determining factor in the causation of dementia paralytica. Gumma of the testicle has been observed after trauma to the organ. Trauma of the skin and soft tissues of a patient in the late stage of syphilis may result in the appearance of any of the clinical forms of late syphilitic lesions. In short, trauma in a syphilitic person may determine the site of syphilitic lesions in almost any organ of the body. Indeed, it appears from clinical observation that syphilitic lesions may develop in areas of the skin that have been traumatized shortly before the patient is infected with syphilis. The appearance of syphilids at the site of tattoo marks may be cited.

Chesney, Turner and Halley [33] furnished experimental proof that trauma determines the site of syphilitic lesions in rabbits. They uniformly observed the appearance of a chancre in the scar of a healed wound after inoculating the rabbit intratesticularly or intravenously.

Consistent with this clinical and experimental evidence, interstitial keratitis may appear after trauma to the cornea.

Trauma as a factor in determining the localization of syphilis in the eye more intimately concerns the cornea as a precipitating cause of interstitial keratitis than any other ocular lesion of syphilis. However, other structures may be concerned, for example, the appearance of a syphilitic iridocyclitis following ocular operations.

In an analysis of 100 cases of interstitial keratitis at Wills Hospital Clinic, the disease appeared following corneal trauma in 3 patients.

The rôle of trauma in the causation of interstitial keratitis has been the subject of much controversy. A number of instances of interstitial keratitis appearing after trauma are reported in the literature. Particular mention may be made of the comprehensive report of Hildebrand [34] and of Höber [35] in which cases are cited and the literature reviewed.

The influence of trauma in the causation of interstitial keratitis will be discussed in more detail in a separate report.

1934 Spruce Street.

33. Chesney, A. M.; Turner, T. B., and Halley, C. R. L.: Studies in Experimental Syphilis: VIII. On the Localization of Syphilitic Lesions in Inflamed Areas, Bull. Johns Hopkins Hosp. **42**:319 (June) 1928.

34. Hildebrand, A.: Ueber die Beziehungen zwischen Trauma und Keratitis interstitialis, Inaug. Diss., Leipzig, 1913.

35. Höber, J.: Ueber Keratitis parenchymatosa nach Verletzung, Beitr. z. Augenh. **8**:102, 1909-1912.

MEASUREMENTS OF RETINAL VESSELS IN EARLY HYPERTENSION *

F. H. HAESSLER, M.D.
AND
THEODORE L. SQUIER, M.D.
MILWAUKEE

In connection with other investigations being carried on at the present time in hypertension, an attempt has been made to demonstrate a measurable change in the caliber of the retinal arteries before and during the administration of amyl nitrite, and to study by actual measurement the relation between the caliber of an artery and that of its associated vein in various degrees of hypertension.

The patients studied were selected entirely on the basis of blood pressure readings from a group of persons who were in sufficiently good health to be carrying on their usual work. Arbitrarily, a systolic level of 140 mm. was set as the upper limit of normal blood pressure, and at the time of the study of the eyegrounds, the blood pressure of the patients so selected was often found to be normal. These patients were, nevertheless, included in this study as a control group. In the group studied, there was probably a wide variety of early hypertensive diseases, in respect to both etiology and type, as only the level of the blood pressure was used as a basis for selection.

TECHNIC

Following dilation of the pupils, the patient was seated in front of the Zeiss-Nordensen camera; blood pressure readings were taken until a constant level was reached, and the minimum pressure was recorded. The fundus of each eye was photographed. An amyl nitrite pearl was then broken and the patient instructed to inhale the fumes, while the head remained in position for photography of the fundus at the moment the signal was given. The blood pressure fluctuations were followed by one of us until at the point of maximum fall in pressure, a photograph was taken and the minimum pressure after amyl nitrite recorded.

Contact prints were made from the negatives so obtained, and corresponding vessels in the same eye before and after the administration of amyl nitrite were marked for measurement. All photographs that were not sufficiently sharp to yield accurate measurements were discarded. Measurements of the blood vessels were made from the positive prints, a Gaertner comparimeter microscope magnifying 32 diameters and sensitive to a reading of 0.001 mm. being used. This degree of sensitivity is such that the accuracy of measurement is limited only by the grain

* Submitted for publication, Sept. 12, 1931.

* From the A. O. Smith Department of Preventive Medicine.

of the photographic emulsion and similar technical factors. Individual measurements varied but slightly, and the mean of three readings was recorded. In some fundi it was possible to select as many as four arteries and veins that seemed to run parallel, and it was assumed that such paired vessels might be taken as representative of the quantitative artery-vein relationship throughout the fundus. Although at the beginning of the study we had planned to measure the diameter of all the arteries and all the veins and compare the sums of the squares as representative of the total area of the cross-sections, a few measurements convinced us that irregularity in balancing of the vessels made this impracticable. Often each of two arterial branches had the same diameter as the stem. Then, too, the smaller vessels that leave the disk substance could not always be identified reliably in the photograph as vein or artery.

RESULTS

Fundus photographs from forty-eight patients were sufficiently clear to make them suitable for measurement. It was found that the caliber of the blood vessels showed tremendous variation, not only from person to person, but also in the same eye. Because of this variability, we believed it advisable to study especially the ratio of the cross-section area of the artery divided by that of its associated vein. Wherever possible, the diameter of a vein peripheral and central to an arterial crossing was also measured before and after the administration of amyl nitrite. The square of the measured diameter of the vessels was used as representing more closely the lumen of the vessel concerned.

In table 1 the results so obtained are arranged in order of increasing systolic blood pressure. Table 2 shows the same results rearranged according to increasing magnitude in the fall in systolic blood pressure after inhalation of the fumes of amyl nitrite. From these tables it is evident that no correlation can be demonstrated in the various characteristics studied.

The data were also grouped in order of magnitude of the percentile increase in the arteries after the administration of amyl nitrite. In table 3 these results are tabulated. No relation could be established between the relative increase in arterial caliber after the inhalation of amyl nitrite and the blood pressure level.

By inspection of the photographs, the cases were then grouped according to the degree of tortuosity present in the retinal vessels, no distinction being made between tortuous arteries and veins because of the small numbers. The results of this grouping are shown in table 4. The average blood pressure was about the same in each of the four groups, classed according to tortuosity as none, slight, moderate and severe, respectively. Furthermore, no appreciable difference could be detected in the percentile change in arterial caliber in these groups after the inhalation of amyl nitrite.

TABLE 1.—*Results of Measurements of Retinal Vessels Arranged in Order of Increasing Systolic Blood Pressure*

Case	Blood Pressure		Degree of Tortuosity Retinal Vessels	Arteriovenous Ratio		Percentile Change in Arterial Caliber After Amyl Nitrite			Peripheral Central Arteriovenous Crossing	
	Before Amyl Nitrite	After Amyl Nitrite		Before Amyl Nitrite	After Amyl Nitrite	Maximum	Minimum	Average	Before	After
950	110/ 88	96/ 70	None	0.50	0.62	28	28	28	1.43	1.07
4728	112/ 80	100/ 56	None	0.72	0.65	0	—36	—18	1.10	1.00
4692	116/ 88	106/ 80	Severe	0.53	0.64	0	0	0	....	....
4507	116/100	98/ ?	Slight	0.51	0.56	43	9	26	....	....
1592	116/ 84	104/ 82	Slight	0.68	0.72	8	—17	—5	1.58	1.60
Average	114/ 88	101/ 72	.....	0.58	0.66	..	..	6	1.37	1.22
1478	120/ 92	104/ 72	None	0.44	0.62	24	11	18	....	....
3154	122/ 82	106/ 58	Slight	0.59	0.57	32	29	31	....	....
759	126/102	104/ 84	Slight	0.61	0.67	9	9	9	....	....
4551	126/ 76	114/ 62	Severe	0.44	0.60	58	0	29	1.50	1.20
2125	128/ 84	78/ 56	None	0.49	0.52	60	0	31	....	....
Average	124/ 87	101/ 66	.....	0.51	0.60	..	..	23	1.50	1.20
4505	130/ 80	94/ 70	Moderate	0.54	0.59	50	0	19	1.07	0.90
4860	132/100	100/ 76	Moderate	0.47	0.60	27	22	25	....	....
914	136/ 96	106/ 70	Slight	0.57	0.56	15	—9	3	1.30	1.30
4630	136/ 90	118/ 82	Slight	0.44	0.56	128	0	56	....	....
4733	136/ 88	110/ 72	Moderate	0.60	0.61	43	—12	23	....	....
4685	138/100	118/ 96	None	0.33	0.52	80	—16	31	1.50	1.00
4277	138/ 94	112/ 72	Moderate	0.53	0.71	80	27	56	1.50	1.40
Average	135/ 92	108/ 77	.....	0.48	0.59	..	..	30	1.34	1.15
4475	140/ 84	112/ 68	None	0.72	0.70	7	0	4	....	....
4642	140/108	?	None	0.66	0.51	—17	—50	—37	....	....
1532	140/ 70	104/ 64	None	0.32	0.38	28	—25	2	....	....
4579	140/ 98	114/ 76	None	0.77	1.24	0	0	0	....	....
4729	140/102	106/ 78	Slight	0.54	0.77	100	37	69	....	....
1046	142/ 96	122/ 86	Slight	0.52	0.55	0	—11	—6	0.88	0.83
146	142/ 80	96/ 78	Slight	0.56	0.53	100	—28	19	....	....
5053	144/ 98	114/ 80	None	0.76	0.66	0	—19	—10	1.50	1.30
4615	144/ 96	104/ 78	None	0.59	0.68	15	15	15	1.04	1.14
4600	146/ 96	110/ 70	Moderate	0.38	0.51	100	67	84	....	....
2946	146/ 84	100/ 72	None	0.71	0.62	43	—50	—4	1.40	0.92
2908	146/ 88	104/ 66	Slight	0.44	0.60	47	47	47	1.31	1.08
4569	148/ 98	96/ 76	None	0.62	0.57	—26	—40	—33	1.85	1.39
Average	143/ 92	107/ 74	.....	0.58	0.64	..	..	12	1.33	1.11
3496	150/114	174/116	Severe	0.48	0.47	108	9	46	....	....
2172	150/104	146/ 86	None	0.42	0.41	21	18	20	1.90	1.50
14	150/110	90/ 74	Moderate	0.86	0.97	9	—8	1	....	....
1018	150/104	140/ 90	Moderate	0.36	0.46	111	33	85	0.95	1.11
2979	152/ 96	124/ 78	Severe	0.33	0.49	80	—36	7	0.91	1.10
4819	154/112	112/ 80	None	0.62	0.73	29	—29	0	1.16	0.78
2823	154/ 90	126/ 94	None	0.53	0.66	120	25	63	....	....
4559	154/100	130/ 76	None	0.55	0.68	50	21	35	....	....
1956	156/100	114/ 74	Moderate	0.53	0.65	116	—20	26	1.38	1.14
2533	156/ 96	122/ 72	None	0.42	0.33	50	14	32	....	....
4652	156/ 92	114/ 72	Severe	0.37	0.36	0	—14	—7	....	....
Average	153/101	127/ 83	.....	0.50	0.56	..	..	28	1.26	1.13
116	160/ 86	126/ 74	Moderate	0.43	0.49	21	0	11	....	....
4933	168/ 90	140/ 70	Slight	0.38	0.52	33	27	30	....	....
Average	164/ 88	133/ 72	.....	0.41	0.51	..	..	26	....	....
65	172/ 92	140/ 74	Slight	0.40	0.49	50	0	17	....	....
4983	172/116	146/106	Severe	0.68	0.57	14	0	7	....	....
1105	178/138	162/ ?	None	0.60	0.56	—12	—42	—22	....	....
Average	174/115	149/ 90	.....	0.54	0.54	..	..	1	....	....
591	190/114	138/ 92	Slight	0.49	0.60	66	0	33	1.08	0.88
4471	200/ 96	164/ 90	None	0.47	0.46	—7	—43	—25	....	....
3494	204/180	190/180	Severe	0.30	0.29	20	20	20	....	....
Average	198/130	164/121	.....	0.42	0.45	..	..	9	1.08	0.88

TABLE 2.—*Relation of Systolic Drop in Blood Pressure After Inhalation of Amyl Nitrite to Measurements of Retinal Vessels*

Cases	Systolic Fall		Mean Arteriovenous Ratio		Percentile Change in Arterial Caliber After Amyl Nitrite
	Range in Mm.	Average in Mm.	Before Amyl Nitrite	After Amyl Nitrite	
13	4-19	13	0.50	0.56	21
12	20-29	25	0.54	0.66	22
11	30-39	34	0.48	0.53	21
7	40-49	43	0.55	0.60	15
4	50-59	53	0.61	0.67	8

TABLE 3.—*Percentile Change in Arterial Caliber After Inhalation of Amyl Nitrite in Relation to Blood Pressure*

Cases	Percentile Increase in Artery		Blood Pressure		
	Variation	Average	Minimum	Maximum	Average
10	37 to —4	—17	112/ 80	200/ 96	148/ 97
10	0 to 9	3	116/ 88	172/116	147/ 96
7	10 to 19	17	120/ 92	172/ 92	144/ 90
7	20 to 29	25	110/ 88	204/180	140/105
7	30 to 39	32	122/ 82	190/114	151/ 95
4	40 to 59	51	136/ 90	150/114	143/ 97
4	60 to 89	75	140/102	154/ 90	148/ 98

TABLE 4.—*Percentile Change in Arterial Caliber After Inhalation of Amyl Nitrite in Relation to Tortuosity of the Retinal Vessels*

Number Cases	Degree of Tortuosity	Percentile Increase in Artery			Blood Pressure		
		Maximum	Minimum	Average	Maximum	Minimum	Average
20	None	63	—33	6	200/ 96	110/88	145/ 91
12	Slight	69	— 6	26	190/114	116/84	145/ 93
9	Moderate	85	1	36	160/ 86	130/80	144/ 95
7	Severe	46	— 7	15	204/108	116/88	154/109

COMMENT

By means of the photographic method used, a relatively accurate measurement of the caliber of the blood vessels of the retina is available. However, by this method we could find no evidence that such measurements are an aid in the recognition of early changes in the retinal vessels associated with hypertension. The marked variability in the arteriovenous ratio in different parts of the same fundus was surprising, especially when one remembers that the ophthalmologist when inspecting the fundus always tries to make an estimate of this relationship. The change in arteriovenous ratio, which is ophthalmoscopically obvious, in all probability represents an actual narrowing of the lumen of the artery, the result of a permanent change in the vessel wall. The delicate variations in the caliber of the vessels that bring about functional adjustment in the vascular supply of the retina are probably not observable.

No correlation could be established between the change in the caliber of retinal arteries after the inhalation of amyl nitrite and the systolic blood pressure, the fall in systolic pressure during inhalation or the degree of retinal arteriosclerosis as indicated by tortuosity in the vessels. From this preliminary survey, it would seem that tortuosity of retinal blood vessels is a manifestation of much greater importance in retinal arteriosclerosis than changes in caliber. A critical study of the development of tortuosity by means of serial photographs of the same eye taken at semi-yearly intervals might thus yield information of considerable importance.

AMBLYOPIA FROM ETHYLHYDROCUPREINE*

CHARLES M. SWAB, M.D.
OMAHA

Ethylhydrocupreine is the most recent of a series of quinine derivatives to be cited for its value in the treatment for pneumonia. That the drug is used quite extensively is shown by Cross [1] who reported observations made in 126 cases of pneumonia in which it was employed as the principal medication. The present use of ethylhydrocupreine was first suggested by Mendel's [2] publication in 1916. Here he stated that it was preferable to other alkaloids of quinine because its rate of absorption could be controlled. As ethylhydrocupreine is soluble in the hydrochloric acid of the stomach, his practice was to give 5 ounces (141.7 Gm.) of milk with each dose of the drug to lessen the concentration of the free hydrochloric acid in the gastric contents. This lengthened the time necessary to convert the base into the acid salt, and prevented its rapid absorption into the blood stream in concentrations sufficiently high to produce symptoms of a toxic character.

That the drug may be productive of toxic effects is realized by the manufacturers, who advise cessation of the treatment in cases in which the patient complains of tinnitus, gastro-intestinal distress or visual disturbance. In reviewing the contributions on this subject, one finds but few recorded instances of impaired visual function resulting from the use of ethylhydrocupreine. The following recent case therefore seems to call for consideration.

REPORT OF A CASE

Mrs. R. B., aged 31, the wife of a physician, was admitted to Immanuel Hospital on Feb. 24, 1931, suffering from a central, lobular pneumonia. She had a temperature of 103.4 F., a pulse rate of 120, 36 respirations per minute, blood-tinged sputum, pain in the central portion of the chest and chills. Later, roentgen examinations revealed an area of infiltration in the root area of the right lung. Sputum typing showed pneumococci, type 4. Urinalysis demonstrated a trace of albumin with both hyaline and granular casts. The blood count exhibited a leukocytosis of 28,000, with 91 per cent polymorphonuclears. These observations are presented to show that the patient was critically ill.

* Submitted for publication, Sept. 23, 1931.

* From the Department of Ophthalmology, Creighton Medical College.

* Read before the Kansas City Society of Ophthalmology and Oto-Laryngology, Kansas City, Mo., Sept. 12, 1931.

1. Cross, F. B.: M. J. & Rec. **126**:271 and 354, 1927.

2. Mendel, F.: Deutsche med. Wchnschr. **42**:537, 1916.

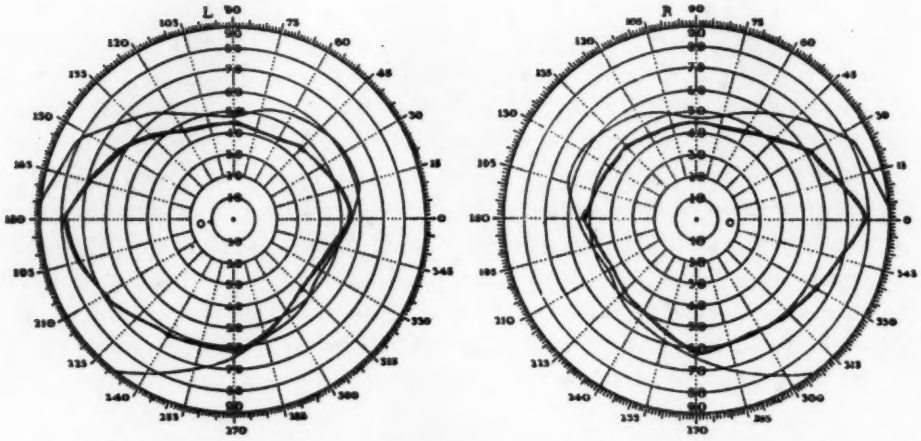


Chart 1.—Field taken in good afternoon daylight on March 5, 1931, with Schweigger bedside perimeter and 2 mm. test object. No color fields were demonstrable with test objects of 2 mm. diameter. The patient was in bed. Uncorrected vision was 6/12 in both eyes.

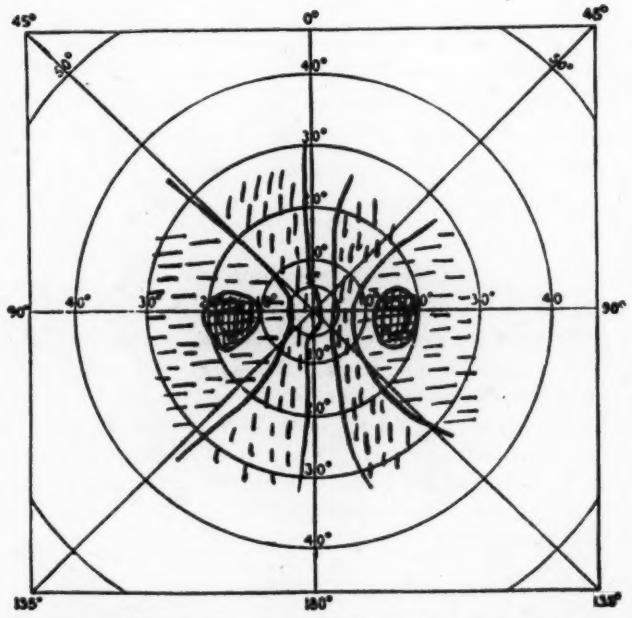


Chart 2.—Cross-hatched areas show field outlined with a 2 mm. white test object; horizontal lines, field outlined with a 4 mm. red test object, and vertical lines, field outlined with a 4 mm. green test object (March 5, 1931).

Ethylhydrocupreine was given in 4 grain (0.259 Gm.) doses every four hours. Mendel's direction that milk be given with each administration of the drug was adhered to; in fact, milk was the only nourishment allowed during this treatment. Although it was given oftener than the ethylhydrocupreine, stimulants and sedatives were not administered by mouth during these two days. The ethylhydrocupreine was continued for ten doses, the last being administered at 8:50 a. m., February 26. About six hours later, the patient complained of inability to see. On that day she was examined by Dr. D. R. Owen, who reported: "Vision reduced to mere light perception; pupils widely dilated and unresponsive to light; pallor of both disks with attenuated arteries. Diagnosis, quinine amaurosis." Strychnine sulphate was then prescribed.

The patient's general condition showed some improvement after the third day, but the visual catastrophe produced a tremendous nervous shock. I saw the patient for the first time on February 28, when the following notes were recorded: She was able to count fingers with each eye at a distance of 1 foot (30.48 cm.). The conjunctivae were pale. The pupils measured from 5 to 6 mm. in diameter. They reacted briskly to light and in convergence and accommodation. Examination of the fundus of the right eye showed: media, clear; disk, oval; axis, 90; slight astigmatic blurring; good color throughout, but with a somewhat lessened capillarity in the temporal portion; the principal branches of the central vein and artery were normal in appearance as to caliber, course and size; in the fovea there was a bright reflex but not an abnormal one; the retina between the fovea and disk was quite ischemic in places, but was not evenly affected; there were restrictions in the arterioles throughout this area, but none was thrombotic. The fundus of the left eye was essentially the same.

Owing to the return of at least some visual acuity, the active pupillary reflexes, the lack of total retinal opacity in the macular regions and the absence of cherry red spots in the maculae, a favorable prognosis was given. In addition to strychnine, amyl nitrite was prescribed. Hot compresses were also ordered, as they were deemed helpful in improving the circulation in the eyes and also of benefit in allaying the patient's nervousness. At this time her mental state was little short of dementia.

On March 5, five days later, vision was 3/6 in each eye without correction. The near point of each eye was 17 cm. The conjunctivae were anemic in appearance. The disks were practically normal in color, and the vessels were entirely normal. There was not the slightest evidence of retinal ischemia. The visual fields exhibited normal peripheral limits with a 2 mm. white disk and a Schweigger hand perimeter. With the same instrument and colored test objects of similar size no color fields were demonstrable. With the Peter campimeter the blind spots were enlarged for white with a 2 mm. test object. With a 4 mm. test object they were greatly enlarged for green and only slightly less so for red.

As improvement continued, the patient was discharged from the hospital on March 9, and returned to her home outside the city. She was next seen on April 27, at my office to which she had come for refraction. The vision of each eye was 6/12 —1. Both near points were 14 cm. The pupils were unequal, the left being larger. Both fundi were normal. Refraction under homatropine cycloplegia showed: in the right eye, with a —1.50 sphere +1.75 cylinder, axis 85, vision of 6/6 —1; in the left eye, with a —1.00 sphere +1.75 cylinder, axis 100, vision of 6/6. A postcycloplegic examination was made on May 1, and correction was prescribed as follows: right eye, —1.75 cylinder, axis 175; left eye, —1.50 sphere +1.75 cylinder, axis 95. With this correction the muscle balance was within nor-

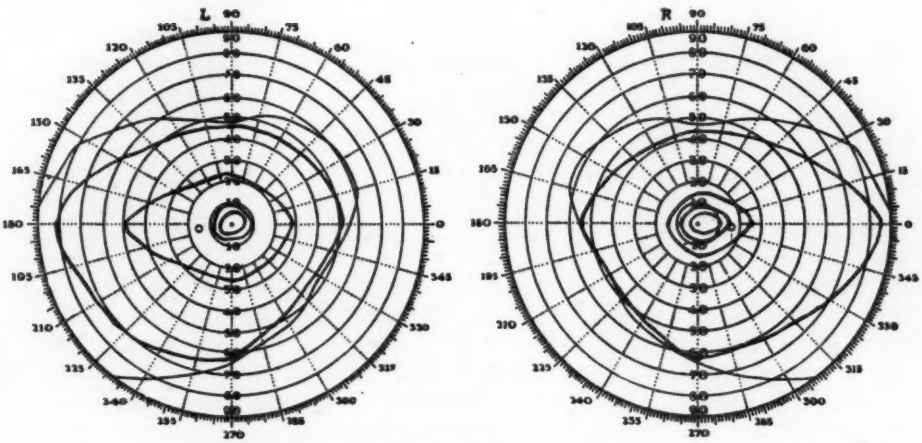


Chart 3.—Fields outlined with 4 mm. test objects on May 1, 1931. The fields for color were in proper relation. Uncorrected vision was 6/12 in both eyes.

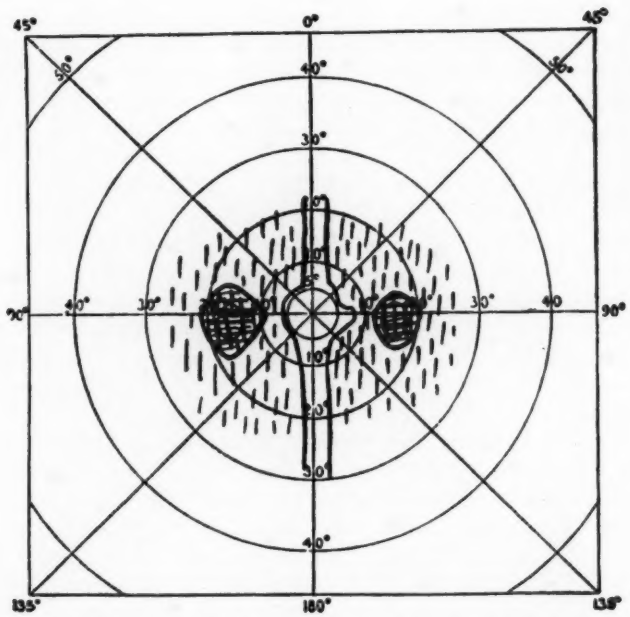


Chart 4.—Fields outlined with a 2 mm. test object on May 1, 1931. A relative scotoma for green was present.

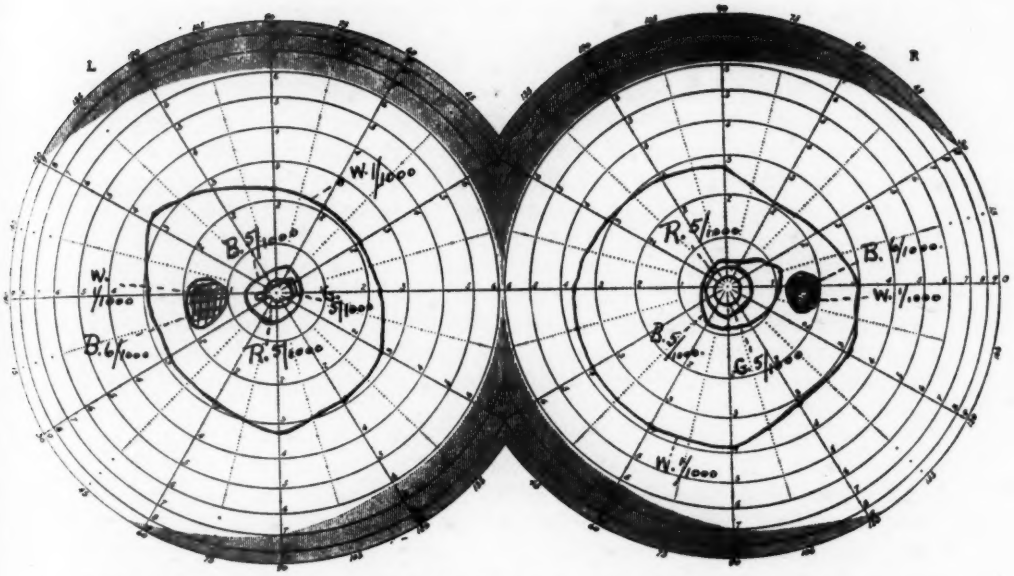


Chart 5.—Tangent screen fields taken at 1 meter with 1, 5 and 6 mm. test objects on May 6, 1931. In the left eye there was interlacing of red and green. Vision was 6/6 in each eye.

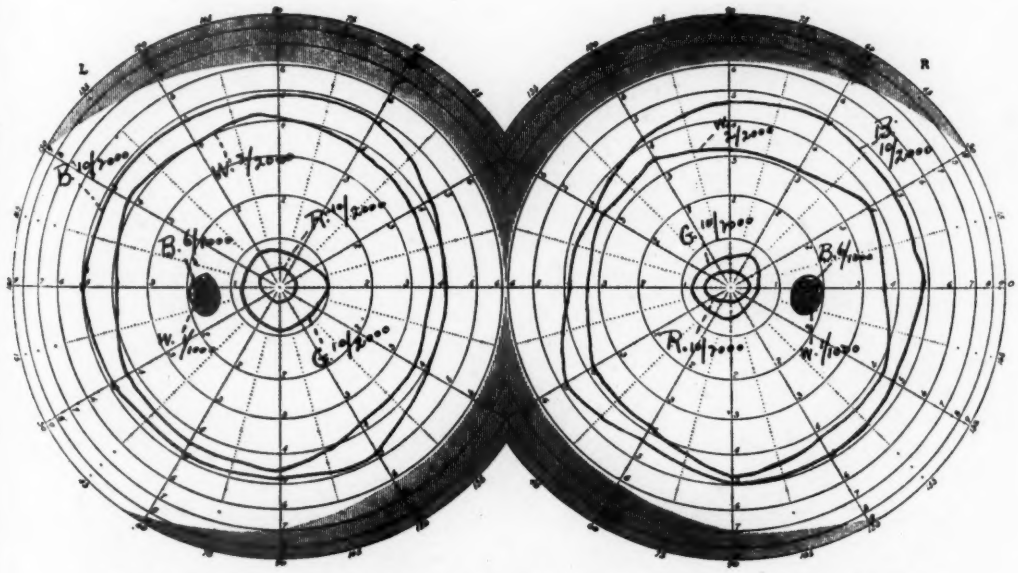


Chart 6.—Field for red inside of field for green with same size test objects at similar distances (Aug. 18, 1931). Note the marked expansion of the field for blue. Vision was 6/6 in each eye.

mal limits. The visual fields were outlined at this time with 4 mm. disks and a McHardy perimeter. The form fields were full in each eye. In the right eye there was contraction for blue, red and green. In the left eye the contraction of the color fields was even greater except in the field for blue, which was of fair extent. The central fields were taken with the campimeter. Both blind spots were enlarged, the left being the larger for the 2 mm. white test object. With the green 2 mm. disk, there was scarcely any central field in either eye. However, there was a small field for green around the point of fixation in each eye.

In order to make a more accurate study of the fields of vision, the patient was requested to return on May 6, at which time quantitative fields were outlined on Walker's tangent screen. The form field was full in each eye, but the color fields were markedly contracted. In the left eye there was interlacing of red and green. The blind spots exhibited only slight enlargement in each instance. Even with this poor grade of color field, Mrs. B. was able to identify any particular color in a small, varicolored, checked dress pattern. She had not the slightest subjective ocular symptom.

Tangent screen fields were again taken on August 18 in order to follow up the case for any possible new development. At this time the fields for blue had become much larger than they were in May. The fields for red and green were greatly contracted, but, strange to say, the greater field of the two was that for green. In fact, the field for red may be said merely to have existed, as it could only be outlined around fixation with a 10 mm. disk at 2 meters. This limited color field, however, was adequate to meet the requirements of this fastidious young woman.

COMMENT

It has been held by some critics that the blindness which occasionally occurs in quinine poisoning is not caused by the quinine alone. Their argument is that, as the drug is usually administered in the treatment for such conditions as malaria, pneumonia or infected abortion, these toxic conditions may have rendered the visual apparatus susceptible to the deleterious effects of quinine. The contention is not without point, although in the American Encyclopedia of Ophthalmology [3] it is stated that the exact clinical picture of quinine amaurosis can be produced in healthy dogs. Furthermore, the experiments of Barabashew [4] on human beings have seemingly answered this objection entirely. By giving toxic doses of quinine to healthy persons who had volunteered for the purpose, he was able to demonstrate all the ocular symptoms of quinine poisoning.

In 1891, de Schweinitz [5] pointed out the following histologic observations based on animal experiments: thickening and changes in the walls of the central vessel (endovasculitis); organization of a clot, the result of thrombosis; widening of the infundibulum of the vessels as the result of the constriction of the surrounding nerve fibers, and,

3. Wood: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1919, vol. 14, p. 10820.

4. Barabashew, P.: Arch. f. Augenh. **23**:91, 1891.

5. de Schweinitz, G. E.: Tr. Am. Ophth. Soc. **6**:23, 1891-1893.

finally, practically complete atrophy of the visual path, including the optic nerve, optic chiasm and optic tracts as far as they could be traced. He also observed that the direct effect of quinine is on the vasomotor centers, producing constriction of the vessels; that changes in the vessels themselves are set up, owing, perhaps, to an endovasculitis; that thrombosis may occur, and that the net result is an extensive atrophy of the visual tract.

Holden,[6] in 1898, dealt largely with the histopathology of the retina and optic pathway of dogs that had been poisoned by quinine. In these experiments the animals were subjected to toxic doses of the drug for from three to forty-one days. By means of advanced staining methods he was able to demonstrate minute changes in the ganglion cells; vacuolation, uneven staining of the chromophilic granules, nuclear displacement and, finally, almost complete disintegration of the entire ganglion cell layer. There was a thinning out, in places, in the nerve fiber layer, and among the fibers were found round globules of a myelin-like character. The optic nerves stained by Marchi's method showed marked degeneration of the myelin sheaths in long-standing cases. Holden concluded that the process in quinine amblyopia is one of degeneration of the nerve fibers and ganglion cell layers of the retina, due, apparently, to insufficient nourishment because of the constriction of the retinal arteries. This degeneration is followed by an ascending atrophy of the optic nerve fibers.

Forster's[7] experiments were made with special reference to ethylhydrocupreine hydrochloride. This drug is familiar to all ophthalmologists for its pneumococcidal properties in pneumococcic conjunctivitis. In his experiments Forster used rabbits, and found that they could tolerate large doses of quinine and ethylhydrocupreine hydrochloride without any apparent toxic effect. He noted that not one of the rabbits exhibited any corneal anesthesia, dilation of the pupils, ischemia of the retina, constriction of the retinal vessels or evidence of visual disturbance. Microscopically, however, he noted perivascular infiltration in the cerebrum and, to a less degree, in the cerebellum. In the retina, the ganglion cell layer showed vacuolation of the cell, protoplasm and nuclei, breaking up of the chromatin, loss of the nuclear wall, a decrease in the number of ganglion cells and the appearance of hyalin-like bodies.

For many years there had been ardent exponents of the quinine treatment for pneumonia. This particular therapy, however, had lost its popularity by 1911. At that time Morgenroth and Levy[8] published an epochal communication which created great interest in ethylhydro-

6. Holden, W. A.: Tr. Am. Ophth. Soc. **8**:405, 1898.

7. Forster, A. E.: Am. J. Ophth. **6**:376, 1923.

8. Morgenroth, J., and Levy, R.: Berl. klin. Wchnschr. **48**:1560, 1911.

cupreine in the treatment for pneumococcic infections. The soluble hydrochloride of ethylhydrocupreine then became the popular quinine remedy. As some writers, however, did not differentiate between ethylhydrocupreine and ethylhydrocupreine hydrochloride, there is no little confusion as to the drug actually employed. At all events, there were many cases reported in which visual impairment was emphasized. Uhthoff [9] published statistics in 1916 to show that visual disturbances occur in from 3 to 4 per cent of patients treated with ethylhydrocupreine. In 1916, Mendel [2] issued his observations on the use of ethylhydrocupreine in pneumonia, controlling the absorption of the drug by giving milk with it as an acid-neutralizing medium. Although one of his patients developed transient amblyopia, there was no visual impairment after twenty-four hours, even when the treatment was continued. In the case of Mendel there is no confusion of terms; he employed ethylhydrocupreine.

From this period ethylhydrocupreine appears to have supplanted all other quinine-like preparations in the treatment for pneumonia. Morgenroth and Schnitzer [10] demonstrated by their experiments that this drug possessed greater pneumococcidal activity than any other quinine product. Baldwin and Rhodes [11] have shown that the pneumococcic properties of ethylhydrocupreine are effective in all four types of the pneumococcus. Smith and Fantus [12] determined that ethylhydrocupreine and quinine are essentially the same by qualitative analysis, but that quantitatively they are different. They expressed the opinion that ethylhydrocupreine is the more toxic.

The large group (126 cases) reported by Cross [1] presented no ocular symptoms, and the smaller series (35 cases) cited by Baldwin and Rhodes [11] exhibited no unfavorable ocular condition. However, there have been a few scattered cases of pronounced visual impairment. Among these is the somewhat recent case of Alvis,[13] in which the patient had not made anything like a satisfactory recovery at the time of his report. In the case presented here, the patient has made an excellent recovery except for restricted color fields for red and green.

The idiosyncrasy of certain persons in their reaction to quinine is also found in other derivatives of cinchona bark. It is therefore difficult to regulate the dosage of these drugs, as they may produce toxic

9. Uhthoff, W.: Klin. Monatsbl. f. Augenh. **57**:14, 1916.

10. Morgenroth, J., and Schnitzer, R.: Ztschr. f. Hyg. u. Infektionskr. **103**: 441, 1924.

11. Baldwin, H. S., and Rhodes, D. R.: U. S. Pub. Health Serv., Hyg. Lab. Bull., no. 149, 1925, p. 69.

12. Smith, M. I., and Fantus, B.: J. Pharmacol. & Exper. Therap. **8**:53, 1916.

13. Alvis, B. Y.: Amaurosis Following Ingestion of Ethylhydrocupreine, Arch. Ophth. **2**:328 (Sept.) 1929.

symptoms even in relatively small quantities. However, it seems only fair to repeat a statement made earlier in this discussion, namely, that few cases of visual impairment from ethylhydrocupreine have been reported.

CONCLUSIONS

1. A patient acquired amblyopia from 40 grains (2.59 Gm.) of ethylhydrocupreine given in accordance with the teaching of Mendel.

2. This is probably an instance of idiosyncrasy.

3. The central visual acuity returned quickly after the withdrawal of ethylhydrocupreine and the substitution of strychnine, amyl nitrite and hot compresses.

4. It may be doubted whether this treatment has any effect on the restoration of vision, but it cannot be denied that it has a beneficial psychologic effect.

5. The form field was not found to be damaged to any extent when enough vision was present to permit of the necessary investigation.

6. Even though there was a complete absence of color perception there was an eventual return of the fields for blue, red and green.

7. The field for blue became full after a period of six months; the field for green showed some increase in size, but was still greatly contracted; the field for red was entirely inside that for green, and was present merely in a small area around fixation.

8. Even with such markedly reduced color fields the patient experienced no subjective symptoms due to this limited color perception.

REFRACTION CHANGES IN THE EYES OF CHILDREN UNDER SIX YEARS OF AGE*

LOUIS BOTHMAN, M.D.
CHICAGO

It has been the common belief among ophthalmologists that the refractive changes in the eyes of children occur in the direction of myopia. As this has not been my impression during routine refraction, it was decided to make a survey of a group of cases in which more than one refraction was done under atropine. The cases reported are from the private practice of Dr. E. V. L. Brown and myself and from the Max Epstein Clinic of the University of Chicago.

There are 124 children in this group, all under 6 years of age. The youngest patient was 10 months old and the oldest 5½ years of age. In 60 per cent of these cases several refractions were done from six to twelve months apart. The final reading was used in this report. The average age of the entire group was 3.95 years when the first determination was made and 5.61 years at the second examination, an average of 1.66 years having elapsed between examinations. Among the group there were 45 boys, or 35.1 per cent, and 79 girls, or 64.9 per cent.

In all cases the eyes were treated for four days, 0.5 per cent solution of atropine sulphate being used in children under 3 years of age and 1 per cent solution in all over that age. Each patient received a minimum of 14 drops of the drug in preparation for the determination of the refractive error. The usual skiascopy was done at a distance of 1 meter. Full correction was ordered except in cases of divergent hyperopia. There were 6 cases of mild atropine poisoning in the series.

The parents of the children studied desired advice because of convergent strabismus in 85 cases, poor vision in 31 and divergent squint in 6. The group contains a very special type of eyes in that it consists of children who may have unusually strong ciliary muscles which did not relax easily under atropine on first examination when the patient had not previously worn glasses.

The group contained 114 cases of hyperopia, 6 of myopia and 4 of mixed astigmatism. Three of the children with myopia showed less

* Submitted for publication, Oct. 13, 1931.

* From the Department of Ophthalmology, the University of Chicago, Dr. E. V. L. Brown, director.

myopia on the second examination. In the 4 cases of mixed astigmatism the eyes remained the same on the subsequent examinations. One patient was emmetropic when first examined and hyperopic later (right eye, + 4; left, + 3.25 diopters). There were three cases of progressive myopia in the group; one child had mixed astigmatism on the first examination and compound myopic astigmatism on the second; the other children were myopic on three examinations. The average increase in the myopia was 1.50 diopters in nineteen months.

Of the cases of convergent squint, there were 44 in which the eyes were parallel with the glasses, 25 cases in which there was less convergence with glasses than without and 16 cases in which the glasses made no difference in the degree of squint. The convergence varied from 5 to 45 degrees, and as many cases in which the eyes were parallel with glasses showed a high degree of squint as those that had only from 5 to 10 degrees of convergence at the onset. The average

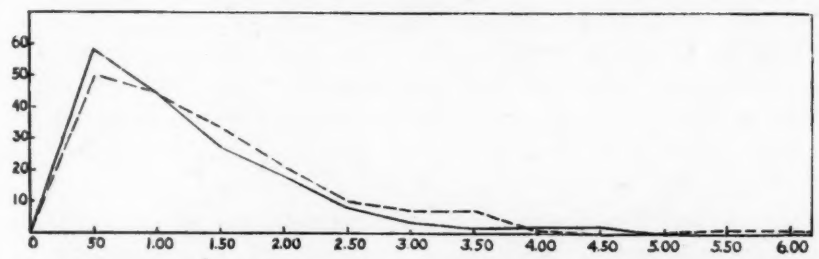


Chart showing increase in spheres. The straight line indicates the spheres; the dotted line, total refractive error; the abscissas, diopters; the ordinates, the number of eyes.

improvement with glasses was 11.9 degrees. The average degree of squint without glasses was 19, and with the full correction (which was always ordered for convergent squint) 4.

Among the group of convergent strabismus there were 13 cases in which there was more convergence (higher degree of squint) without glasses after they had been worn for a year or more than there was without glasses when the eyes were first examined.

The following refractive changes were observed in all the eyes of the 124 cases: Of these, there were 178 eyes that showed an increase and 38 a decrease in the amount of spheres, while 32 remained unchanged. Of the total refractive error of these eyes, 178 were increased, 31 decreased and 31 remained unchanged. The average increase in the sphere was 1.07 diopters and in the total error (sphere plus cylinder) 1.28 diopters. For the 38 cases in which the weaker meridian decreased, the average was 0.64 diopter, and for the 31 eyes in which the stronger meridian decreased, the decrease averaged 0.75 diopter.

A chart is presented that shows only the increase. The base line represents the first refractive error and the dotted line the increase in the total refractive error or the stronger meridian, while the straight line represents the increase in the weaker or spherical meridian only. It may be seen at a glance that these two curves are very similar, that the sphere increased most often under 1 diopter and that the total error increased more beyond that power.

In the eyes in which there was no squint, 65 per cent showed an increase, the average increase being 1.00 diopter in the weaker and 1.50 diopters in the stronger meridian. There were 25 per cent, including the cases of progressive myopia, which showed an average decrease of 0.87 diopter in the weaker and 1.20 diopters in the stronger meridian. Ten per cent showed no change in the sphere and 5 per cent no change in the cylinder. These figures are similar to those for the entire series and, it would seem, indicate that the eyes with less hyperopia show approximately the same changes as those with higher degrees of hyperopia, namely, eyes with convergent strabismus.

It is true that this series is a small one from which to draw undisputed conclusions, but since 178, or 71 per cent, of the 248 eyes showed an increase in both meridians on the second determination of the refractive error under atropine, it would seem that in this type of hyperopic eye in patients wearing the full correction there may be an increase in the hyperopia up to 6 years of age, and not a decrease as is commonly believed to be the case.

Clinical Notes

CATARACT AND A MODIFIED CATARACT KNIFE*

FASSETT EDWARDS, M.D., OAKLAND, CALIF.

In the excellent company of hundreds of other operators, for a long time I have been seeking some betterment in the technic of the always formidable operation for cataract.

In casting about for improvement, the various instruments have been considered, rather than the method of doing the work, although that likewise has been given much thought.

It so happens that not many are given the unique opportunity of being the originator of anything radically and specifically new. Only a very few now have anything new to offer ophthalmologists. But many of this profession try—and succeed—at improvements of details that may seem trifling and of little worth, yet the aggregate of all the betterments eventually means almost a complete revamping of the operative procedure in question.

The speculum as used in cataract work is far from being the best instrument obtainable, and I have in mind some ideas on which I hope to be able to work, when time shall allow. However, one does worry along somehow with what one has. Yet if one were inclined to doubt the inefficiency of the present operating speculum, a study of the many differing types would be quite convincing of such dissatisfaction.

The relative value of a cystotome and the capsule forceps have been discussed endlessly; and those who believe in one or in the other instrument continue to think about as they did before. Personally—and such matters are even more personal than the type of razor one employs in the morning struggle with the ever recurring beard—I prefer the cystotome for the reason that it seems, in my hands, to result in bringing less dangerous pressure on the anterior capsule during operation. (I always suspect fluid vitreous, and sometimes am not disappointed.) In order to grasp a wet, rounded surface, it seems to be almost an instinctive feeling that one must press somewhat. And "somewhat" may be quite too much. The plan which in my own work has been fairly satisfactory has been to use a cystotome with a rather long, very sharp blade, set on a very thin, flexible shaft. After the section is made, this cystotome is inserted on the flat to the lower edge of the anterior capsule, turned upright and a cut attempted in a circular form about the edge of the capsule.

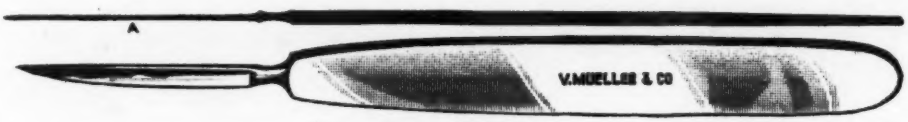
This procedure, by its delicate encircling pressure, seems to break, in whole or in part, the zonule of Zinn, so that often I have the lens in its capsule to slip smoothly out through the corneal section, or the lens alone will emerge. I do not always get the same result because I am

* Submitted for publication, Oct. 26, 1931.

not able to determine the thickness or resistance of the anterior capsule, and of necessity must make about a certain amount of pressure—within what seems to me to be the limits of safety.

So far as I am concerned, I seem to get less resulting trouble and have quite as good end-results when I make a full half-corneal section. I have used all sorts of superior flaps, but I have reached the conclusion that I only make my work harder and get no better results than I now obtain from an incision entirely in the clear cornea.

But, possibly as a matter of my own personality, knives have, up to recently, been a source of considerable dissatisfaction to me. They have not pleased in this or in that respect. Some months ago I cast about for an improvement in the standard von Graefe cataract knife, and pored over many catalogues. Eventually I came across the knife originally devised by Dr. Frank Wilson of Bridgeport, Conn., which was further changed by Colonel Smith of India. Its blade seemed, mechanically, to be shaped perfectly to enter the anterior chamber with a minimum amount of pressure, and at the same time to have sufficient stiffness to afford one a sense of dependability, although it did then seem that the blade was overlarge, probably because prior to that time I had been using knives with very short, narrow blades.



Modified cataract knife.

Getting one of the Smith-Wilson knives, I tried its blade. The blade was all I could want; I was greatly pleased. But the 5 mm. square, aluminum handle always gave me a sense of insecurity, a feeling that it would perhaps roll about in my fingers, so the thought naturally followed: Why not correct that apparent defect by making the handle broad and flat, as well as adding slightly to its weight by using steel as a material?

I sent a sketch of my idea to V. Mueller & Company, Chicago, and asked them to make a sample knife. They made two. Herewith is shown a cut of the second and better one. The upper view shows the back edge of the knife, the lower one its side. The cut is in full size.

This knife is made of a single piece of the finest steel, which is finally chrome plated, except that the blade itself is crocus polished. Of course, such a knife blade should not carry plating of any kind.

My personal manner of holding this knife is to lay it across the fingers near their tips, with the thumb opposing at about the center of the flat handle. Because of its flat handle and its weight, the knife gives one a feeling of being anatomically a part of one's hand—adherent, yet free. Nor does it need to be grasped with any but the most delicate touch. It seems to cling easily to the fingers.

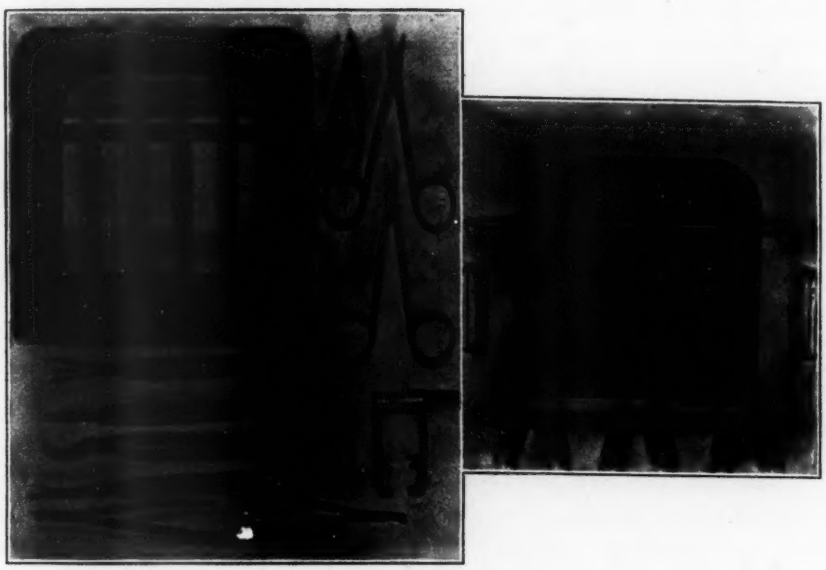
I have used this knife with all satisfaction in a number of cases, and have passed it on to my friends for study, although I think that none

of them has put it to test by operation. Some of them held it in their hands and said that they did not like it. It does feel quite different. However, so intimate a tool as one's cataract knife is a matter of personal liking; and with this thought the knife is offered to the public for such judgment as it may see fit to pass.

A METAL INSTRUMENT RACK

R. TOWNLEY PATON, M.D., BALTIMORE

In order to facilitate the handling of sharp instruments, the metal rack shown in the illustrations was designed and found practical. The



Rack to hold sharp instruments.

instruments once placed in the rack need not be touched until the operator or his assistant removes them at the time of operation. Repeated handling of the sharp instruments during the process of sterilization may thus be avoided.

Better protection is afforded when a large number of instruments are placed in the same tray. Sufficient clearance is allowed so that the knives when removed by a forward sliding motion do not come in contact with the edge of the basin.

Various sized cataract knives, keratomes, discission needles and capsulotomes will fit this rack.

---

* Submitted for publication, Nov. 5, 1931.
* From the Wilmer Institute, Johns Hopkins Hospital.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Propaganda for Prevention of Blindness.—The International Association for the Prevention of Blindness, Paris, France, has issued a journal entitled "L'animateur des temps nouveaux," under the editorship of M. Louis Forest. In this booklet are a number of terse statements on the value of eyesight, the manner in which the eyes work, the stresses that various types of work involve, and the risks from careless, dirty, and wilful habits which are often directly accountable for blindness. Each page deals with one subject, and each subject is illustrated by one or more sketches. Even the type has been made to serve a demonstrative purpose, making the chief points on the page stand out sharply. This journal is a most effective piece of propaganda, perhaps the best of its kind that we have seen. (From *Brit. M. J.* **1:** 159 [Jan. 22] 1932.)

UNIVERSITY NEWS

Dr. Morie F. Weymann, of Los Angeles, has been appointed associate professor and acting head of the department of clinical ophthalmology in the medical school of the University of Southern California. Appointments of associate clinical professor have been given to Drs. A. Ray Irvine, Theodore Lyster, Clifford Walker, A. L. Macleish, Carl Fisher and George McCoy.

SOCIETY NEWS

Chicago Ophthalmological Society.—At the annual meeting of the Chicago Ophthalmological Society on January 18, the guest speaker, Dr. Benedict, of Rochester, Minn., spoke on "Orbital Tumors." The paper was discussed by Dr. G. deTakáts, of the department of surgery of Northwestern University. At the annual election, Dr. Michael Goldenburg was elected president, Dr. Wright Williams vice-president and Dr. Richard Gamble secretary-treasurer.

American Academy of Ophthalmology and Otolaryngology.—The Academy will hold its annual meeting in Montreal, Canada, Sept. 19 to 23, 1932.

Obituary

GEORGE S. DERBY, M.D.
1875-1931

George S. Derby died on Dec. 12, 1931, of pneumonia, after a short illness. Born in Boston, May 29, 1875, the son of Dr. Hasket Derby, one of the first and most eminent ophthalmologists in New England, he received his medical education at the Harvard Medical school and as intern in the Massachusetts General Hospital. He then went abroad to prepare himself for his specialty, spending most of his time in Freiburg under Professor Axenfeld and with Professor Fuchs in Vienna. On his return to Boston in 1903, he became identified with the Massachusetts Charitable Eye and Ear Hospital, and succeeded his father on the service at the Carney Hospital in Boston, which position he occupied for ten years.

Dr. Derby saw extensive service in the late war from 1917 as member of Base Hospital No. 5, stationed with the British; he then became attached to the headquarters of medical and surgical consultants A. E. F., and was discharged in 1918 with the rank of lieutenant-colonel, receiving the highest recognition for meritorious work as consultant in ophthalmology.

On resuming his work in Boston in 1919, his field of activity rapidly enlarged, and his sense of leadership manifested itself. His career from this time on is a simple record of increasing responsibility and resulting honors. He was asked to organize the Infant's Hospital, which he successfully did, so that later it was taken over by the Children's Hospital; he also started a wetnurse directory in Boston. He acted as secretary of the Section on Ophthalmology of the American Medical Association for many years, and was an important factor in the development of this Section, until it has become the largest and most influential organization of ophthalmologists in America. He was chairman of this Section in 1923-1924.

He became professor of ophthalmology at the Harvard Medical School in 1924, and reorganized the ophthalmologic service at the Massachusetts Eye and Ear Hospital. He was the first to appreciate the importance of a social service in eye hospitals, and established a department at the Massachusetts Eye and Ear Hospital, which is now serving as a model for other hospitals and is conducting a training school for social service workers.

Dr. Derby was the commanding figure in ophthalmology in New England, and the interests of the New England Ophthalmological Society, of which he had been president, were always foremost in his mind. He was president of the Suffolk Medical Society at the time of his death.

He became one of the editors of the ARCHIVES OF OPHTHALMOLOGY, after it was taken over by the American Medical Association, in 1928; he was interested and active in the National Society for the Prevention of Blindness, and was actively at work developing the Howe Ophthalmological Laboratory at the time of his untimely death. Whatever he

GEORGE S. DERBY
1875-1931

became interested in, he bent every effort to advance, working for its best interests, and his strength was shown by the fortitude with which he pursued the goal that he had set.

In addition to all of these responsibilities, and the care of a large private practice, Dr. Derby was a faithful attendant at medical meetings, where, by papers and discussions, he was always ready to further the interests of ophthalmology. He was a forceful speaker, and his contributions to debate were always arresting and his opinions always commanded respect. His contributions embrace all subjects in ophthalmology; he was interested particularly in studies of the light-sense in glaucoma, which were carried out with a special apparatus.

In 1916, the Knapp Medal of the Section on Ophthalmology of the American Medical Association was bestowed on him for his paper on "Some Aspects of Ophthalmia Neonatorum." He was a pioneer in the spread of information about the early stages of glaucoma and how to care for it, and he had a great interest in ocular neuroses.

And now this busy life is suddenly cut off. The many enterprises that he helped will go on, thanks to his efforts, and the many young men who were fortunate enough to be associated with him owe him a great debt for his wonderful example.

He will be greatly missed not only by the wide circle of his personal friends, but also by the large circle of those of the same specialty, at whose meetings he has been for so many years a familiar figure.

And yet when a friend thinks of George Derby, it is at first on his outstanding personal qualities rather than on his professional success that he naturally dwells. The motive force in his life was service, and the key to his character was loyalty. Of George Derby's personal qualities, it is a privilege to speak. His character was one of singular beauty; he was never known to say an unkind word to any one or of any one. His modesty, his reticence when it came to talking of oneself, his personal charm and kindness of heart endeared him to all with whom he came in contact. He had a serenity of disposition, an influence exerted by a deep religious conviction in his every act and thought. His piety was manifest in his consideration for others, his modesty, his kindliness and in his large-minded and large-hearted character.

For the many who today feel bereft, his work, his understanding heart, his wise counsel and his ever present friendship will be an abiding memory.

Dr. Derby is survived by a devoted wife, Mary Brewster Brown, of Portland, Maine, who was his faithful helpmate, and by two children, Hasket Derby and Mary B. Derby, for whom great sympathy is felt.

A. K.

It is difficult for me adequately to express on paper what were my feelings for George Derby, they were such a mixture of respect, of admiration and of affection.

As a colleague, finding myself continually trespassing on his special field of work, I came to lean on him heavily for advice, and never failed to get immediate help, however great the sacrifice of time.

Contact with him as a fellow practitioner was an inspiration. He radiated something that promptly instilled confidence on the part of every patient, and this inevitably grew into a strong attachment for him. Whatsoever pressure he may have been under, he never gave the slightest hint of being hurried.

He was a man of high emotional nature, which he kept under such perfect control it was unapparent. Modest to a degree and apparently unaware of his own attainments, his life was one of service to his beloved branch of the profession, to his school and to his community. He was from every standpoint a well-nigh perfect human being.

HARVEY CUSHING.

George Derby became known to English ophthalmic surgeons in the early years of 1900 when he worked at Moorfields, but it was during the War—when he came to France in the Harvard Unit—that by his strong personality, his power as a worker, his selflessness and his charm as a companion, he became widely known and loved.

When the American Army came to France he was appointed consulting ophthalmic surgeon to the Army in conjunction with Dr. Greenwood, and did brilliant work both as an organizer and as a clinician. Before this he worked as ophthalmic specialist for the British Army, both in the Harvard Unit and also at the English Ophthalmic Center at Boulogne, rendering very valuable service. There was never a task so trivial but he did it with alacrity, nor was there a problem so difficult but he would whole-heartedly discuss and throw new light on it. All who worked with him were aware of his high ideal of service, and though he was extremely reticent those who knew him well appreciated that this was based on deep religious conviction.

In later years at the English Speaking Ophthalmic Convention in London, and at the International Ophthalmological Congress in Holland, he, with his genial cheery face and great broad shoulders, was constantly a center to which many were drawn by his personal magnetism.

He was a man of sterling uprightness and of great sympathy. His judgments both of people and in his professional work were wise and kindly, enlivened by a happy sense of humor, and it was almost unknown for him to make a disparaging criticism of any one.

The loss of George Derby will be deeply felt both in his own country and in England by a large circle of acquaintances, while to his many friends his death leaves a blank which it is impossible to fill.

W. T. LISTER.

George Derby became secretary of the Section on Ophthalmology of the American Medical Association in 1913. He was reluctant to accept the position, his modesty causing him to underate his ability to fill it. Once elected, however, he took up the duties of the office with zest and filled the position most acceptably until he was chosen chairman of the Section some years later.

Being thus brought into contact and correspondence with ophthalmologists all over the country, Dr. Derby soon developed a national interest in our specialty which later on was extended still further by his activities in the American Ophthalmological Society, in which organization he had a hereditary interest by reason of his father, Hasket Derby, having been one of its founders and most active workers.

George Derby missed but few meetings of these societies and took an active part in the scientific business of both. In these meetings his splendid figure and dignified bearing gave weight and carried conviction to his carefully considered utterances.

He had been well trained in ophthalmology, and as his touching obituary to Professor Axenfeld, which appeared in the ARCHIVES, testifies, was especially appreciative of the teaching he had received from that revered master. While a student in Freiburg, he had prepared his first paper on ophthalmology and published it in Professor Axenfeld's journal, the *Klinische Monatsblätter für Augenheilkunde*. The International Congress held in Amsterdam in 1929 gave Dr. Derby an opportunity to renew his association with Professor Axenfeld, and with his family to revisit the Bavarian Highlands, which he had grown in student days to love so well. For George Derby was a great lover of nature and had the physique to find entertainment and relaxation in all forms of outdoor sports.

His summer home in Maine, on the estate of his wife's family not far out of Portland, on the shores of Casco Bay, gave him ample opportunity for boating in which he and his family took keen delight. His was a lovable nature, and those fortunate enough to have seen something of his home life could not but be impressed by the beauty of his family relationship. There was much of the spiritual in George Derby, which increased his love of outdoors and his friendship for his fellow man. This love of his fellows, this interest in mankind, was the key to his success in life and made him broadminded and eager to help others. It made him a great clinician and developed in him the power of organization so that quietly but successfully he amalgamated the ophthalmic interests in Boston into a great center of clinical practice, teaching and research. No one could have effected this great movement more modestly than he. Had he lived, he had other plans in conjunction with the Howe Foundation which would have made Boston a still greater center.

Dr. Derby's broad sympathies led him to take an interest in the National Society for the Prevention of Blindness. As a member of its board of managers he took personal and active interest in the welfare of that organization, and he inaugurated the movement of placing medical social workers who had been trained in ophthalmic work in the

Massachusetts Eye Infirmary in hospitals in other cities. By his writing he did much to instruct laymen regarding the prevalence and dangers of glaucoma.

His friends will miss his counsel and cooperation. Steadfast and forceful, he combined these sterling qualities with the gentleness, kindness and humor that made it a delight to be with him. An able organizer, a skilful operator, an able writer and speaker, in fine, he was an ideal figure physically, mentally and spiritually. The future held much for him. There was no eminence to which he might not have attained, no honor which his colleagues would not willingly have bestowed upon him.

WILLIAM CAMPBELL POSEY.

Correspondence

GUIST EYE SPECULUM

To the Editor.—The Guist eye speculum is a very great aid in operative procedures, especially in such operations as require section of the globe to any extent.

There is, however, one feature of its application that adds danger in cataract operations. If the instrument is adjusted in such manner as greatly to lift the eyelids forward, with consequent pulling tension on the conjunctival folds, especially when a superior rectus stay suture is used, there is a marked tendency to collapse and recession of the anterior contents of the globe after section has been made. In other words, the lifting puts a pulling tension on the periphery of the globe forward from the equator and is likely to cause sinking backward of the lens and iris margin deeper into the globe immediately following the section, thus making it more difficult, or indeed impossible, to deliver the lens or do iridectomy.

I believe that this warning should be brought to the attention of operators who are beginning the use of the Guist eye speculum.

V. A. CHAPMAN, M.D., Milwaukee.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

A PECULIAR FORM OF COLOBOMA OF THE CHOROID. B. A. SOCOLOVA, Russk. j. opht. **8:** 347 (March) 1931.

The author describes the case of a patient who had a typical coloboma of the choroid and optic nerve of the right eye, the vision being unimpaired. In the left eye he had only 0.07 vision, not improved by correction. The optic nerve was normal. The choroid presented a very unusual picture, a triple coloboma in three sections with normal strips of fundus between the defects of the choroid. The iris of both eyes had no coloboma.

O. SITCHEVSKA.

Bacteriology and Serology

BACTERIOLOGIC RESEARCH ON TRACHOMA IN ALGERIA. V. MORAX, Rev. internat. du trachome **8:** 133 (July) 1931.

Because of the author's failure to find Bacterium granulosis of Noguchi in cases of trachoma seen in France, it was thought that if cases were examined when fresh and untreated more likelihood of obtaining the organism would be present. However, in spite of using the methods of the Rockefeller Institute, Morax was unable to isolate the organism in Algeria.

L. L. MAYER.

ABSORPTION OF LENS REMNANTS FROM AN OPERATION FOR CATARACT AFTER A CATARACT EXTRACTION ON THE OTHER EYE. INJECTION OF LENS ANTIGEN. FRANCISCO PEREZ ALLENDE, Arch. de oftal., Buenos Aires **6:** 411 (July) 1931.

Having observed a renewed absorption of lens remnants from an operation for cataract in a young woman, following the extraction of a cataract in the other eye, the author, after some experimentation, decided to prepare emulsions of lens nuclei to be injected in cases in which soft lens matter is left behind after extractions of cataracts. With some exceptions this seemed to promote a more rapid absorption.

He uses one lens nucleus for 2 cc. of physiologic solution of sodium chloride, which he places in two 2 cc. ampules. He makes the first injection into the deltoid region, and injects one ampule daily.

The author finally reviews the literature on the subject of the serologic properties of the crystalline lens.

C. E. FINLAY.

Color Sense

MONOCHROMATIC VISION. B. CHANCE, Am. J. Ophth. **14:** 417 (May) 1931.

The author reviews the literature and discusses the etiology and symptoms and the different explanations advanced concerning mono-

chromatic vision. It is suggested that achromatopsia should not be regarded as a division of color blindness. A large bibliography is appended.

W. S. REESE.

Conjunctiva

OCULAR PEMPHIGUS AND SKIN GRAFTS. F. TERRIEN, GOUGEROT AND HASSON, Arch. d'opht. **48:** 275 (April) 1931.

The authors' case of classic cutaneous pemphigus, with predilection for the mucous membrane of the eyes and mouth, is especially interesting from the point of view of treatment. Although it is often thought that treatment is of no avail, recently suggested methods have yielded unhoped for results. These are autohemotherapy, sodium cacodylate and the arsenobenzenes and intramuscular injections of tartrobismuthate of sodium. More recently Spillman and Watim have recommended the subcutaneous injection of from 5 to 10 cc. of a 5 per cent Witte's peptone solution, 40 drops of epinephrine being given by mouth the first day, 0.2 Gm. of ergotin orally the second day and an intravenous injection of 5 cc. of a 10 per cent solution of calcium chloride the third day. The authors point out the necessity of having several modes of treatment available, as in some instances one form may aggravate the condition in one patient and another in others. The observation by the patient of the cessation of the cutaneous lesions following an attack of measles with high fever led the authors to use autohemotherapy to produce a temperature reaction, with success in their case.

The prevention of symblepharon in these cases is almost impossible. In the authors' case they successfully dissected the lids from the globe and introduced skin grafts. This resulted in greatly improved motility of the eyes and the relief of pain and photophobia. A patient with a similar case was successfully operated on in 1928.

S. B. MARLOW.

PNEUMOCOCCUS CONJUNCTIVITIS WITH ULCERS OF THE CONJUNCTIVA. G. TIRELLI, Arch. di ottal. **38:** 243, 1931.

In a case of acute conjunctivitis, the author observed a number of yellowish areas of ulceration in the upper fold. These were superficial and from 5 by 3 mm. to 3 by 2 mm. in size. The submaxillary glands were swollen. A smaller ulcer was present on the bulbar conjunctiva and another in the skin of the lower lid. Pneumococci were found in scrapings and cultures from the ulcers. Under treatment with ethylhydrocuprein ointment the conjunctivitis cleared up rapidly. After five days the ulcers were much swollen and six days later were healed. The cornea was never involved. The patient was a veterinary surgeon and the possibility of some other infection from his professional duties was considered, but the rapid improvement under ethylhydrocuprein seemed to confirm the diagnosis of a pure pneumococcus infection. Enlargement of the glands is rare, and ulcers of the conjunctiva, according to the author, have never been described in pneumococcus conjunctivitis. This complication may have been due to lack of care and unusual exposure to wind and dust during the first days of the infection.

S. R. GIFFORD.

Congenital Anomalies

THE DEVELOPMENT OF CONGENITAL CORNEAL OPACITIES. RUDOLPH HOFFMANN, Arch. f. Augenh. **105:** 162, 1931.

Two main causes have been considered for the development of congenital opacities in the cornea, (1) inflammatory processes during fetal life and (2) developmental anomalies. The majority of recent opinions have been in favor of developmental defects. Peters and others consider anomalies of the lens to be the primary factor, whereas Seefelder holds the view that the primary cause is a defect in the endothelium of the cornea. The author presents a case that supports Peters' point of view.

A résumé of the history follows: A 5 year old girl had corneal opacities noted since birth in both eyes. One brother had congenital defects consisting of opacities in the lens capsules and marked pupillary membranes. The other children and the mother had normal eyes. In the patient under consideration, the right cornea was clear save for one spot where there was a dense opacity against which the lens was plastered. The latter was clear with the exception of its point of contact with the cornea. The corneal endothelium was present throughout the cornea except at the point where the lens was fastened. There was an incomplete atypical coloboma of the iris. No changes could be seen in the vitreous or in the fundus. The left eye was similar to the right with the addition of a partial dislocation of the lens.

The author concludes that the corneal opacities must certainly have been due to a congenital developmental anomaly, since there were no signs of any previous inflammation and since there were other evidences of developmental anomalies present, i. e., the coloboma of the iris. He considers it probable that there was a defect in the cutting off of the lens from the ectoderm, and that hence the endothelium never developed in this spot.

F. H. ADLER.

Cornea and Sclera

SCLERITIS AS A SEQUEL OF HERPES OPHTHALMICUS. G. G. PENMAN, Brit. J. Ophth. **15:** 585 (Oct.) 1931.

In four of the cases of herpes ophthalmicus seen by the author, scleritis developed later. In three of these, it was of a characteristic type; in the fourth, a sclerokeratitis. He describes a typical case as follows: About two to three months after an attack of herpes ophthalmicus, usually accompanied by some iridocyclitis, the patient complains of redness and pain in the affected eye. On examination, one or more nodules of a dark red color and about the size of a lentil are found beneath the conjunctiva, which is much injected and has a glossy surface. The nodules are slow in subsiding, usually taking months to do so, gradually getting smaller and darker, almost plum-colored, and finally disappearing, leaving sharply defined slaty-gray areas in the sclera where they have been situated. The conjunctiva over these areas is freely movable and apparently normal. The iris is atrophic, often in patches, which are found in that part of the iris nearest to the slaty areas in the sclera. The pupil often remains quite inactive and dilated. The con-

dition is liable to relapse, and fresh nodules may arise in areas of the sclera hitherto uninvolved. The great majority of the cases occur in persons over 50 years of age. In addition to the characteristic condition described, in which there is no concurrent corneal involvement, a sclerokeratitis sometimes occurs.

W. ZENTMAYER.

EPISCLERAL TUBERCULOSIS. P. SCOTTI, Ann. di ottal. e clin. ocul. **59:** 430, 1931.

In a woman aged 61, six weeks after a slight abrasion of the left eye, a fleshy growth surrounding the outer limbus developed. This was firmly attached to the sclera, extended 1 cm. from the limbus and was surrounded by intense congestion of the bulbar conjunctiva. The patient was poorly nourished, and died two years later of pulmonary tuberculosis. Four months after she was first seen by the author, the mass had surrounded the limbus and was broken down on the surface, and a corneal ulcer was present. An attempt was made to remove the mass, but it was found to extend deeply into the sclera, and the eye was enucleated on account of severe pain.

Sections showed the mass to be composed of tuberculous granulation tissue containing numerous giant cells. This tissue had extended through the sclera at the limbus, invading the root of the ciliary body and the suprachoroidal space. The cornea was involved near the outer limbus and had broken down on its surface. No caseation or necrosis was present in the mass itself.

Unusual in this case, as compared with cases of conjunctival tuberculosis, was the rapidly progressive course with involvement of the sclera, and this also differentiated it from the rare infections of the globe with Actinomyces and the molds. A few atypical cases of tuberculosis that somewhat resembled the present case are reviewed. None of these showed the symptoms of severe pain present in the author's case. While an exogenous infection is considered to be the cause in most cases of conjunctival tuberculosis, the present case can best be explained as an infection through the blood stream, and this is probably true of most cases of episcleral tuberculosis, the infection being aided in some cases by trauma.

S. R. GIFFORD.

KERATOCONUS. D. D'AMICO, Ann. di ottal. e clin. ocul. **59:** 456, 1931.

Various theories of etiology are reviewed, and the author concludes that while general factors, such as glandular imbalance, may play a part in some cases of keratoconus, they are absent in many others, and a local cause, such as deficient resistance of the central cornea, must always be present.

For the optical correction of the condition, the author has tried contact glasses, but without notable success. He reports a case in which he carried out Cirincione's procedure, cauterizing the cornea just below the apex of the cone so as to form a semilunar eschar with its concavity at the lower border of the pupil. This was repeated twice, the operation causing marked flattening of the cone each time; the cone returned to its previous condition, however, after about ten days. At a fourth

intervention the cornea was perforated with the cautery, the chamber remaining empty for ten days. Two months later the scar was tattooed with india ink, and vision of 2/10 was obtained, which has persisted for two years. Perforation of the cornea is apparently necessary for the success of the method, the chamber being allowed to remain empty for at least a week.

S. R. GIFFORD.

THE CLINICAL IMPORTANCE OF THE PERMEABILITY OF THE CORNEA. F. P. FISCHER, Klin. Monatsbl. f. Augenh. **86**: 298 (March) 1931.

The cornea is the main port of entrance into the globe for solutions dropped into the conjunctival sac. Nine tenths of the solution enters through the cornea and one-tenth only through the conjunctiva, lymph and blood vessels. The cornea owes its permeability to the condition of the epithelium and endothelium. The corneal stroma is fairly indifferent; when deprived of either the outer or the inner epithelial layer, it will swell by contact with the aqueous humor or tears and offer no resistance to passing solutions. Epithelium and endothelium, on the other hand, always prevent such passage, but to a varying extent in different directions, depending on the degree of swelling. The less the epithelium is swollen, the less fluorescein will stain. Anatomically intact, the epithelium may, occasionally, stain vividly if the degree of swelling is increased; other substances, however, under these circumstances, will permeate less actively. The endothelium, which is far less inclined to swell than the epithelium, will swell if the sodium chloride in the anterior chamber is diminished; hence the greater protection offered the corneal stroma by the endothelium. Water permeates the intact cornea more readily from the outside; sodium chloride and oxygen enter the anterior chamber readily; carbonic acid passes out of the eye only from the anterior chamber.

The permeability of the cornea is reduced in tuberculous iridocyclitis, preventing or retarding the action of mydriatics. The subconjunctival injection of mydriatics in very small quantities, in such cases, will bring about the desired result. The quantity of atropine, scopolamine, physostigmine and pilocarpine that enters the anterior chamber is not proportionate to the concentration of these drugs, as they are instilled into the conjunctival sac. The permeability depends on the hydrogen concentration of these solutions. Pilocarpine hydrochloride solution of p_H 4 or basic atropine, scopolamine and physostigmine of p_H 7.5 will enter more readily. A formula for atropine is added:

℞ Atropine nitrate..... 0.1
Sodium bicarbonate... 0.03
Sodium chloride..... 0.04
Water to make.....10.00, to be used fresh or in ampules.

Nitrates are superior to sulphates, corresponding to the rapidity of diffusion, which is greater in univalent ions.

Anesthetics failed to increase the permeability of the cornea. Fischer recommends the use of eye drops at a temperature of 30 C.

K. L. STOLL.

CALCIUM THERAPY IN PHLYCTENULAR DISEASE OF THE EYE. M. CREMER, München. med. Wchnschr. **78:** 1223 (July 17) 1931.

A series of 150 cases of phlyctenular disease in children was divided into two groups. The same general care was given to the patients in both groups, but in one group they were given calcium gluconate by mouth, in addition. The average duration of the condition in calcium-treated patients was 14.1 days, while the average in the noncalcium-treated group was 22.5 days. During a period between 9 and 22 months later, about 100 of these children were reexamined. Relapses occurred in only 8 per cent of those having received calcium therapy, while 14 per cent of relapses were found in the other group. The author believes that calcium therapy is indicated in phlyctenular keratitis as it shortens the acute period and militates against relapses to a greater degree than other methods.

L. L. MAYER.

General

BRIEF STATEMENT CONCERNING ONCHOCERCOSIS IN MEXICO. R. SILVA, Am. J. Ophth. **14:** 518 (June) 1931.

In 1925, in Mexico, news was received of a disease in the state of Chiapas which produced blindness. This was presumed and later proved to be onchocerciasis. This disease was also found in the mountainous region of the state of Oaxaca. A commission found 15,000 cases in Chiapas and 5,000 in Oaxaca. The responsible organism was Onchocera volvulus-caecutiens. This disease is characterized by subcutaneous tumors in the hairy skin and more rarely in the remainder of the body; mild elephantiasis of the skin of the face, which assumes an olive green tinge, and ocular manifestations in the form of punctate keratitis and plastic iritis, causing seclusion and occlusion of the pupil. Penetration of the eye by microfilarias was demonstrated. Transmission was found to be through Simulium demnosum, mooseri and ochraceum. Dispensaries were opened, and treatment was instituted, which consisted in extirpation of the cysts. Four sanitary brigades were organized, which operated during the dry season, and the proprietors of coffee and farming estates were instructed to prevent the procreation of carriers by screening windows, cleaning up ditches, etc. Pamphlets telling of the dangers and means of preventing the disease were sent out.

W. S. REESE.

General Diseases

LOCALIZATION OF INTRA-OCULAR TUBERCULOSIS AND JUVENILE ANGIOPATHY. T. AXENFELD, Klin. Monatsbl. f. Augenh. **85:** 465 (Oct.) 1930.

This paper has as its chief subject the group localization of hematogenous tuberculosis of the eye. Recurring phlyctenular scrofulous keratoconjunctivitis in Europe is doubtlessly connected with tuberculosis. The idea of its origin by pyogenic surface inoculation or by "exudative diathesis" may be refuted. "Eye scrofulosis" develops in the form of phlyctenas and allied corneal diseases only when the tuberculosis has

invaded the bronchial glands. It is a process of allergy. Exogenous irritations and various bacteria may complicate the condition. The virus of tuberculosis is the deciding factor, even though this can hardly be demonstrated by culture, inoculation or staining methods. It would be a step backward, in Axenfeld's mind, if the connection between recurring phlyctenular keratoconjunctivitis and "scrofula," i. e., tuberculosis, would not be duly considered. He points out, furthermore, that the destructive tuberculosis of other organs seems to be less frequent in those persons who have suffered from an early scrofulosis, and that they seem to be protected against actual intra-ocular tuberculosis. Intra-ocular tuberculosis is usually not found in patients with pulmonary, or surgical, tuberculosis, nor in those afflicted with lupus, but in people who present no tuberculous habitus. Tuberculin tests and roentgenograms of the thorax showed merely constant changes in the bronchial glands and an occasional involvement of the lungs. One group seems to exclude the other. On the other hand, miliary tuberculosis of the lungs is frequently complicated by the manifestation of miliary tubercles in the choroid. This fact rendered the diagnosis difficult until it became known that the larger number of all inflammations of the iris, the ciliary body and, also, the choroid and the adjoining sclera are of a tuberculous nature. Stock's experiments were valuable in clearing this question. He proved the susceptibility of the uvea to tubercle bacilli. Axenfeld admits that focal infection may occur, but by exact and continuous observation on persons with this type of uveitis, the tuberculous condition can usually be demonstrated.

Tuberculous retinitis was never observed by Stock and others; it is as rare as the formation of tubercles proper in the retina. Tuberculous diseases of the retina and the disk may lead to occlusion of the terminal retinal vessels and to extended necrosis.

Juvenile angiopathy, or tuberculous retinal phlebitis, however, is a typical retinal disease, characterized by recurring hemorrhages. Axenfeld, in 1909, called it a hemorrhagic form of ocular tuberculosis. It seems to be identical with the "recurring hemorrhages in the vitreous" of young persons. The uvea is not involved at first. The tubercle bacilli do not get into the retina by way of the blood circulation, but in all probability they enter the perivenous lymph sheaths after leaving the intact capillaries. This would suggest that tubercle bacilli may be in the tissues of the body without causing any disease. Axenfeld then compares this retinal periphlebitis with that of the brain.

K. L. STOLL.

Hygiene, Sociology, Education and History

PHYSICAL FITNESS ESSENTIAL TO DRIVING AN AUTOMOBILE. H. COPPEZ, Arch. d'opht. **48:** 254 (April) 1931.

Coppez draws attention to the many recent discussions on the physical requirements, especially visual fitness, necessary for the safe driving of automobiles. He points out that the early proposals as to the regulation of physical fitness were based purely on theoretical grounds and were principally inspired by Roche in 1907. Many changes have taken place since then, not only in the increase in the number of

automobiles and in their greater mechanical perfection for safety and reliability, but also in the improved conditions of the roads from the point of view of their widening and posting for direction and warning against dangerous situations. He discusses the proposed measures of control under the headings of professional, nonprofessional and one-eyed drivers. He believes that there is justification for regulation regarding the physical fitness of professional drivers, but not for all others. Under certain conditions one-eyed persons can be given licenses. He does not think that the preliminary statement in regard to the physical defects of applicants is to be recommended, because it would be difficult to carry out and would lead to unfair consequences.

S. B. MARLOW.

Injuries

A SEQUEL OF ALKALI BURNS NOT PREVIOUSLY REPORTED. OSCAR THIES, Arch. f. Augenh. 105: 47, 1931.

Two cases are described of alkali burns of the conjunctiva and cornea in which ulcer developed several months after the healing of the original injury. The author describes the differences between alkali and acid burns of the eye and points out that the fundamental difference consists in the nature of the eschar. Acids precipitate protein and form a dense, hard eschar, which prevents the penetration of the acid deep into the tissues. Alkalis, on the other hand, dissolve proteins and form a soft eschar, through which the alkali continues to penetrate, reaching deeper and deeper into the tissues.

In the first case reported, that of a lime burn, healing took place in the usual time, but several months later the tissues broke down, forming a stubborn ulcer. This was rapidly healed by covering with a mucous membrane graft. The second case, that of a burn from ammonia, healed in the usual time, and it was not until nearly one year later that the scar tissue in the lower culdesac broke down into an open ulcer. Mucous membrane grafts did not succeed in healing the ulcer, which after many months closed by granulation tissue.

F. H. ADLER.

Lacrimal Apparatus

PRIMARY TUBERCULOSIS OF THE LACRIMAL SAC. M. DEL DUCA, Riv. oto-neuro-oftal. 8: 114 (March-April) 1931.

The author reports a case of a young man presenting a primary tuberculosis of the lacrimal sac. Rapid examination led to the diagnosis of peridacryocystitis. His attention was drawn to three important facts: the existence of purulent fusion of the retromandibular lymphatic ganglion of the same side; the presence of a soft, elastic, pseudofluctuating tumefaction, which could not be reduced even by prolonged pressure and which was perfectly painless, and the perfect permeability of the lacrimal ducts.

The suspicion of tuberculosis of the lacrimal sac was confirmed by the laboratory examination for the tubercle bacillus, found in the pus of the retromandibular gland by inoculating this pus into two guinea-pigs

and by the histologic data of the excised tissues. These tissues showed an intensive infiltration of lymphocytes with numerous epithelial cells and large giant cells.

The absence of lesions in the nose and the sinuses about the nose, in the skin of the face and in the conjunctiva, also the complete absence of any tuberculous lesion in the personal or family histories, lead the author to conclude that he had before him a primary form in which the nose represents the entrance for the bacillus. He admits that the tubercle bacillus, which often remains a long time on the nasal mucosa without proliferation, may penetrate into the nasolacrimal canal and from here into the sac, and lodges itself in the folds of its mucosa. From here it passes on to the corium and to the tissues about the sac owing to the predilection of these bacilli for connective tissues rather than for epithelium.

According to the author, this explains the swelling around the sac with all the characteristics of a peridacryocystitis and the perfect permeability of the lacrimal duct.

V. R. SYRACUSE.

THE ETIOLOGY AND PATHOGENESIS OF DACRYOCYSTITIS. A. B. POLIAK, Russk. j. opht. **8:** 305 (March) 1931.

The author examined the flora of the lacrimal secretion of forty-four patients by taking smears and making cultures on serum-agar. Pneumococci were found in twenty-eight patients, or in 63.5 per cent. In order to determine the virulence, he infected the visceral cavity of white mice with pus taken from the sac and with pure pneumococcic culture. Of nineteen strains of pneumococci, four were avirulent, twelve proved to be virulent but slightly, and three strains were markedly virulent.

Since dacryocystitis is considered a secondary infection to rhinopathy, the author had the nose and sinuses of all his patients examined thoroughly. Sixty per cent of the patients suffered from atrophic rhinitis, the process not always being on the same side as the lacrimal infection. In some of the cases the inflammation of the sac proved to be a familial disease. This fact led the author to believe that at times the nasal dystrophy is secondary to the diseased lacrimal sac.

O. SITCHEVSKA.

Lens

THE BARRAQUER CATARACT OPERATION (MODIFIED). O. R. WOLFE, Am. J. Ophth. **14:** 510 (June) 1931.

The author gives a brief history of the erisiphake method. The author's operating team consists of five members. A preliminary iridectomy is usually done a week or ten days before the extraction. A heavy control suture is inserted beneath the superior rectus. A broad conjunctival flap is obtained, and a suture inserted. The lens is then grasped and rocked, and extraction is aided by pressure at the lower limbus. The suture is then pulled taut, and the eye is closed for from ten to fifteen minutes. In obstreperous patients, a conjunctival flap is

made prior to the section. The author comments on the operation and gives a brief analysis of his results.

W. S. REESE.

ETIOLOGY AND CHEMICAL NATURE OF CATARACTOUS LENSES. P. W. SALIT, Am. J. Ophth. **14:** 523 (June) 1931.

The author takes up "glassblower's cataract," and discusses its appearance and the influence of infra-red rays in its production. Electric cataracts are next referred to, and then cataracts of endogenous origin, beginning with congenital cataract. The influence of thyroid and parathyroid and the rôle of tetany are discussed, and various experimental works are cited. Avitaminosis and rupture of the lens capsule are also referred to as causes of cataract. The relationship between cataracts and sugar tolerance and the coincidentally taken blood analyses are discussed as well as organ specificity of lens protein and the results of lens antigen treatment for cataract. The article is concluded with a discussion of the chemistry of the normal and the cataractous lens.

W. S. REESE.

HEREDITY AND SENILE CATARACT. E. MORETTI, Arch. di ottal. **38:** 288, 1931.

The author reviews previous reports on the heredity of senile and other forms of cataract. In previous cases the female sex has been most affected. In some families all the women aged 50 or more were affected, while in no family with cataract has the male sex alone been involved.

The phenomenon of anticipation, by which succeeding generations are affected at an earlier age, is shown in a number of families with cataract, according to Groenow.

Moretti reports a family in which, among twenty-eight persons, eleven cases of senile cataract occurred in four generations. The cataracts became mature in all cases at about the age of 60.

Since only one parent was affected, and the condition was transmitted directly, it must be considered a dominant characteristic. The incidence of 40 per cent, though lower than called for by Mendel's law, is explained by the death of many members of the family before the age of 60. Seven men and four women were affected, and either sex transmitted the condition directly. No factor other than heredity was found to account for the condition in any case.

S. R. GIFFORD.

Lids

ETIOLOGY OF BLEPHARITIS. A. MEERHOFF, Arch. de oftal., Buenos Aires **6:** 545 (June) 1931.

From an exhaustive analysis of the conditions underlying the chronicity of blepharitis and its resistance to different local remedial measures, the author concludes that primary blepharitis is produced by Koch bacillary toxins, and in treatment he had employed injections of tuberculin after the usual preliminary precautions.

He reports seven cases in which this treatment was used with marked benefit to the patient.

C. E. FINLAY.

Methods of Examination

NEUTRALIZATION IN DYNAMIC RETINOSCOPY. J. I. PASCAL, Brit. J. Ophth. **15:** 589 (Oct.) 1931.

The author's method of procedure is as follows: A retinoscopy is made by instructing the patient, who is already wearing his statically found correction, to fix binocularly the letters on a small chart attached just above the mirror of the retinoscope. The retinoscope is held in the median line at an equal distance from the two eyes of the patient, and each eye is rapidly and alternately "refracted." Assume the test to be made at a distance of 13 inches (32.5 cm.), necessitating a normal convergence of three binocular meter angles and 3 diopters of accommodation. If the initial movement is "with," and for each eye plus 1.0 diopter (added to the distance correction) produces neutralization, then as one proceeds with stronger plus lenses neutralization continues until with a plus 2.5 diopter lens a definite against movement is obtained. The first lens that produced neutralization was a lens of plus 1.5 diopter. This gives a low neutral of plus 1.0 diopter, a high neutral of plus 2.25 diopter, the strongest lens before reversal, and a neutral range of plus 1.25 diopter. The low neutral represents accommodation depending on the size of type used in the test. The inefficiency may be due to exhaustion incident to general bodily weakness, or it may be due to local conditions such as beginning paresis.

W. ZENTMAYER.

UNIVERSAL APPARATUS FOR ROENTGENOGRAPHIC EXAMINATION OF THE OPTIC FORAMEN. R. ARGAÑARAZ, Arch. de oftal., Buenos Aires **6:** 265 (May) 1931.

After emphasizing the importance of determining the size and relation of the optic foramen in the diagnosis of different processes and conditions, the author describes, as a substitute for Goalwin's cap, an apparatus that has for its object the proper inclination of the x-ray tube. The instrument consists mainly of two graduated semicircles fixed at the skull vertex and tragus, respectively, by means of two crossed metal or celluloid bands, such as are used for sustaining head mirrors, to the center of each of which is attached a weighted string, which readily marks the 38 degrees of sagittal and horizontal inclination, which Goalwin finds is approximately necessary for the direction of the ray to coincide with the optic canal.

For determining the diameter of the canal, the author uses Goalwin's formula: $x : d - \frac{c}{d} (f + d)$, in which x indicates the diameter of the canal; d, the diameter of the canal on the plate; a, the distance of the plate from the Coolidge tube; c, the distance of the optic canal to the plate, and f, the diameter of the focal area of the x-ray tube.

C. E. FINLAY.

Neurology

ARGYLL ROBERTSON PUPILS, TRUE AND FALSE. W. J. ADIE, Brit. M. J. **2:** 136 (July 25) 1931.

The true Argyll Robertson phenomenon is an infallible sign of syphilis, but it is important to know exactly what is meant by this term.

Argyll Robertson, in 1869, published in the *Edinburgh Medical Journal* a short article entitled "Four Cases of Spinal Miosis with Remarks on the Action of Light on the Pupil," stating that though the retina was quite sensitive, the pupils contracted at once during active accommodation for near objects, uninfluenced by the amount of light admitted to the eye, and that slow and only partial dilatation ensued on the application of a strong atropine solution. Pupils conforming in every particular to this description are peculiar to syphilis, but it is important to remember the exact definition. Miosis is a pupil not exceeding 2½ mm. in diameter. The retina is sensitive, which means that the vision is good. The pupil contracts at once and fully on convergence. The amount of light admitted to the eye does not influence the size of the pupil. Mydriatics cause a slow and only partial dilatation.

A partial Argyll Robertson pupil is one in which the response to light, direct or consensual, in one or both eyes, is slow and incomplete and in which the other criteria are all fulfilled. Then this partial sign has the same value as the complete phenomenon, but only if the vision is good.

The true Argyll Robertson pupil has never been seen in encephalitis, nor is the pupillary change in quadrigeminal tumors of this character. In the latter condition, the pupils are rarely small, dilate in the dark as well as with mydriatics, and react imperfectly on convergence. Usually there are an associated palsy of conjugate upward ocular movement. papilledema and other symptoms pointing to the presence of a tumor.

Pupils that react neither to light, direct or consensual, nor on convergence are said to be fixed, and in fixed pupils the defects are sometimes partial. Then the reaction to light is more affected than the reaction on convergence.

The myotonic pupil is frequently mistaken for an Argyll Robertson pupil because it does not react to light with the ordinary tests, but does react on convergence. The myotonic pupil is distinguished by its peculiar behavior during convergence, and it is not very rare. It is often unilateral; it is then almost always larger than its fellow. It may be oval with the long axis horizontal or vertical. With ordinary methods, the reaction to light appears to be abolished. After a sojourn in a dark room, however, the pupil dilates, and on subsequent exposure to daylight contracts very slowly. During active accommodation, contraction of the pupil, sometimes after a short delay, proceeds slowly through a range often greatly in excess of the normal. It differs from the true Argyll Robertson sign because it is often unilateral; the pupils vary in size from time to time and are never miotic; the reaction to light though absent to ordinary tests is not abolished; the pupil dilates in the dark, but the reaction on convergence is peculiar; prompt and full dilatation occurs with mydriatics. The cause of this phenomenon is unknown. The iris of the myotonic pupil is unparalyzed, and if the stimulant is intense enough or prolonged, the pupil contracts. Accommodation may be slow, but the range is normal. The author thinks that the term nonsyphilitic Argyll Robertson pupil, as suggested by Foster Moore, should not be used, as it is a contradiction in terms. Myotonic pupil is probably the best name. It is important because it gives rise to an unfounded suspicion of syphilis.

A. KNAPP.

Orbit, Eyeball and Accessory Sinuses

DIFFUSE AND PROGRESSIVE ANGIOMA OF THE ORBIT: DIAGNOSTIC AND OPERATIVE DIFFICULTIES. H. TRUC and C. DÉJEAN, Arch. d'opht. **48:** 290 (April) 1931.

The unusual features of the case reported by the authors are considered by them to be the enormous degree of exophthalmos, the difficulty of diagnosis and the operative risk due to diffuse uncontrollable hemorrhage. The exophthalmos, although it produced no local disturbance and was apparently due to a benign tumor, disfigured the patient so much that she requested operation in spite of the risks. The diagnosis of the nature of the cause was difficult. No pulsation was felt. Pulsation, but no bruit, was heard faintly with the stethoscope. Exploratory puncture was not done. Ophthalmoscopic examination revealed a normal fundus. Vision, as well as the field, was normal. An angiomatous tumor was excised through a curved incision along the superior internal margin of the orbit. Hemostasis was very difficult and resulted in the destruction of the internal rectus muscle. The final result was satisfactory, except for the divergence due to injury of the internal rectus muscle and a slight amount of exophthalmos. Histologic study showed the tumor to be a simple telangiectatic angioma.

The authors suggest that the electric knife would be helpful in the surgical treatment of another case of this kind.

S. B. MARLOW.

HYDATID CYST OF THE ORBIT. SOLARES, Arch. d'opht. **48:** 298 (April) 1931.

Before describing his personal observations, the author calls attention to the work of Teulieres, who collected 141 cases from the literature. He points out the frequently negative Weinberg-Parvu reaction, the lack of eosinophilia and the possibility of an absence of scolices in the sterile fluid of the tumor, and expresses the belief that the inflammatory lesions of the optic nerve sometimes seen in these cases represent a neurotrophic reaction due to a toxin produced by the cyst. After describing his case in detail, he calls attention to the rapidity of the evolution of some of the symptoms. Diplopia developed between fifteen and twenty days after the onset of the first symptoms of pain and a sense of fulness in the orbit. Exophthalmos appeared after a month. The diplopia disappeared at about the time optic atrophy was well advanced, but the displacement of the eye remained unchanged. He was able to extirpate the cyst under local anesthesia. The fluid from it contained scolices and hooks.

S. B. MARLOW.

Pharmacology

TOXIC INFLAMMATORY ACTION OF SOME OF THE ALKALOIDS USED IN THE EYE. F. POOS, Klin. Monatsbl f. Augenh. **86:** 171 (Feb.) 1931.

Referring to his paper on pharmacotoxic reactions of an inflammatory type in the eye (*Verhandl. d. deutsch. ophth. Gesellsch.*, 1930, p. 122), the author adds recent results of his research. The changes produced

by the toxic action of a number of remedies commonly used in ophthalmology are shown histologically. The walls of the blood vessels are chiefly attacked, producing changes that can hardly be differentiated from genuine acute inflammatory conditions. Poos found that eserine, pilocarpine, atropine, scopolamine, homatropine, ergotamine and also the substances employed in local anesthesia, if used outside the globe in large doses, produce extensive intra-ocular changes that can be demonstrated histologically. This toxic action, aside from the desired and well known effect on the function of the nerves and muscles, is shown furthermore in regard to the use of erythrophleine. This drug decreases the intra-ocular tension, as pointed out by Ascher, producing, however, vivid exudation and infiltration, resembling acute panophthalmitis. Cocaine, when instilled repeatedly, causes dilatation of the vessels, edema and migration of leukocytes in the conjunctival sac. This toxic effect may result in atony of the conjunctival, scleral and ciliary vessels and in increased permeability of their walls; leukocytes were found in great numbers in the aqueous humor and the vitreous; the ciliary processes were edematous. Microscopic pictures of rabbits' eyes demonstrate these changes and similar ones produced by atropine. The author recommends weaker solutions of erythrophleine than hitherto used in glaucoma, from 0.01 to 0.02 per cent as a maximum. Cocaine should not be used in intrabulbar operations in doses exceeding those required for complete anesthesia, because hemorrhages may result during or after the operation. This may happen particularly in old people after the second instillation of a 10 per cent solution of cocaine, the toxic dose. Subconjunctival injection of 1 drop of the same concentration near the upper limbus of an inflamed eye, or in the presence of synechiae, is risky; it may lead to hemorrhages into the anterior chamber after the incision.

K. L. STOLL.

Physiology

THE AUTONOMIC NERVOUS SYSTEM. K. KURE, Oration at Annual Meeting of the Japanese Ophthalmological Society, June, 1931, p. 39.

A. General Review.—The autonomic nervous system consists of the sympathetic and parasympathetic nervous systems.

The organs supplied by the autonomic system are innervated both by the sympathetic and parasympathetic, and these two groups are generally antagonistic. Under a central influence they are both stimulated to a definite degree which constitutes their tonus.

It is often difficult to decide which of the two innervations of the organ is at fault. It frequently occurs that both have an increased tonus.

The mode of action of the autonomic nerves differs according to the organ. Some organs are stimulated by the sympathetic and retarded by the parasympathetic, while in other groups the condition is exactly the opposite.

B. Specific Poisons for Autonomic Nerves.—Epinephrine stimulates the sympathetic nerve, increases vascular and cardial contraction, acts on the heat center and increases carbohydrate metabolism.

Tetrahydronaphthylamine stimulates the sympathetic center and the nerve endings.

Ergotoxine-ergotamine paralyzes the stimulating sympathetic branches without affecting the retarding branches.

Pilocarpine, muscarine and acetylcholine stimulate the parasympathetic.

Atropine and scopolamine paralyze the parasympathetic nerves.

Nicotine interrupts the connection between the preganglionic and postganglionic nerve fibers of the autonomic system.

Apocodeine, in large quantities, paralyzes both autonomic systems.

Potassium increases the sensitivity for pilocarpine.

Calcium increases the action of epinephrine. If the nutritive fluids do not contain calcium, epinephrine causes arrest of the heart action in dilatation.

Acetylcholine, neurine and pituitary, in nutritive fluids rich in calcium, produce arrest of the heart action in contraction.

Histamine, pituitary, thyroidin and insulin are poisons for autonomic nerves.

Epinephrine, choline, pituitary, thyroidin and insulin are formed in the animal body. They stimulate the sympathetic and parasympathetic nerves, and regulate the functions necessary for life. In this sense, they are of greater importance than the other poisons. Recent investigations have shown that choline is closely related to the parasympathetic nervous system.

C. Sympathetic and Parasympathetic Innervation of the Eye.—1. The oculopupillary phenomenon consists of a contraction of the pupil, enophthalmos and slight ptosis, and occurs in lesions of the cervical sympathetic or of its center in the spinal column. If the upper cervical sympathetic ganglion is resected a definite time after the section of the cervical sympathetic, the pupil still further contracts. If in animals the stellate ganglion is excised some time after resection of the upper cervical sympathetic ganglion, the miotic pupil becomes still more contracted. This can be explained by the fact that certain fibers pass along the vertebral artery from the stellate ganglion to the pupil.

2. Between the sclera and the choroid there are autonomic nerve plexuses. They are also found in the ciliary body, and are believed to innervate the ciliary muscle and the blood vessels. The iris contains nerve fibers. The pupillary sphincter is innnervated by the parasympathetic, while the dilatator is innervated by the sympathetic.

3. Dr. Shiraishi has experimented with the nutritive innervation of the ciliary ganglion. If the ciliary ganglion on the one side is crushed, the cornea often sloughs away, and the other eye shows a marked corneal opacity which heals after perforation has taken place. These changes are not inflammatory and must be regarded as trophic in nature.

4. After removal of the ciliary ganglion, a dystrophy of the external ocular muscles results. This was also observed after a resection of the cervical sympathetic, and more marked after resection on both sides.

5. It has been seen that the nerve for lacrimation leaves the brain with the facial nerve as the great superficial petrosal to enter the sphenopalatine ganglion terminating in the cells of this ganglion and its postganglionic fibers, passing in the fifth nerve to the lacrimal gland. Recent investigations have shown that there is another group of fibers, which,

as preganglionic fibers, leave the brain with the fifth nerve, terminate in the cells of the sphenopalatine ganglion, return to the fifth nerve and enter the lacrimal gland to cause lacrimation. A. KNAPP.

Refraction and Accommodation

CONTACT GLASSES. A. RUGG-GUNN, Brit. J. Ophth. **15:** 549 (Oct.) 1931.

This is an exhaustive study of present knowledge of the use of contact glasses. The paper opens with a historical record, describes the Zeiss ground glasses and the Müller blown contact glasses, gives full instructions for prescribing and fitting ground glasses, and enters into a discussion of the optical theories involved. As to their use, various factors complicate the issue. The psychology of a people: the author believes that the British people will prove to be on the whole unfavorable. A contact glass, owing to its position in the orbit, is protected from injury much better than a spectacle lens. It will require a much more extended use of contact glasses before it is known what percentage of patients can wear them all day without discomfort. Undoubtedly many persons do. On the other hand, a fair percentage of persons appear to be unable to tolerate them for more than from three to six hours daily even after prolonged trial. They confer on an ametrope an appreciation of perspective and a judgment of distance comparable to that of an emmetrope. By securing uniformity of the size of retinal images in anisometropia, by neutralizing conical astigmatism, and by permitting a full range of movement of the eyes, they completely satisfy practically all the ocular conditions of binocular vision. There is also an increased field of vision. The author feels that they will enter largely into sport. The use of contact glasses in the treatment for certain diseases of the eye as suggested by Heine is stated. In conical cornea and in irregular corneal astigmatism their value is manifest. W. ZENTMAYER.

CONTACT GLASSES OR CROSSED CYLINDERS FOR HIGH ASTIGMATISM. J. H. BEAUMONT, Brit. M. J. **1:** 1068 (June 20) 1931.

Contact glasses are not as uncomfortable as one might imagine. They are applied by the surgeon, using a rubber sucker attached to the glass shell, the concavity of which contains saline fluid. Holocaine is used as an anesthetic. The patient's head is bent forward, the eyelids are held apart, and the contact glass is fitted onto the eyeball, care being taken to avoid air bubbles. At first they should be worn for only an hour a day; later, for a longer length of time, though not at night. They may be used in conical cornea, corneal opacities, high astigmatism, high hypermetropia and high myopia and after extraction of cataract. They are of great value in certain corneal conditions, especially when glasses are not available or cannot be worn.

The author speaks of a case of crossed cylinders in which a +8 cylinder at 90 degrees and a —8 cylinder at 180 degrees were combined. This gave the patient a vision of 6/6, but when this was transposed to —8 +16c at 90 degrees, the vision fell to 6/36. The crossed cylinders, however, caused so much distortion that they were not satisfactory. This difficulty was obviated by the use of a contact glass. A. KNAPP.

INFANTILE MYOPIA. A. BRÜCKNER and A. FRANCESCHETTI, Arch. f. Augenh. **105:** 1, 1931.

The authors describe twenty-five cases of high myopia in childhood, which they separate as a distinct type from the myopia that increases during school age. They term the condition in the cases under consideration "infantile myopia." Two thirds of the cases occurred in boys and only one third in girls. All the children were about the beginning of the school age, i. e., around 6 to 7 years old. They showed a myopia that ranged from 3 to 25 diopters and a myopic astigmatism ranging from 1 to 4 diopters.

The following ophthalmoscopic picture they regard as typical: The papilla is regularly oval and bordered on the temporal side by a small conus, which seldom exceeds more than a third of the diameter of the papilla. Occasionally, beyond this, there is an area of peripapillary atrophy of the pigment epithelium. The pigment epithelium is largely absent between the papilla and the macula and, in fact, throughout the whole posterior pole of the eye. The pigment in the macular region, however, is increased, so that a dark pigmented spot is characteristically found. The retinal reflexes are characterized by their absence, and the blood vessels show little light streak.

The visual acuity of these eyes was never normal even with the best correction, and as one would suppose, the higher the myopia the poorer the vision. The visual fields and the dark adaptation were within normal limits.

A distinct hereditary influence could be traced in only a few of the cases. For the most part, the parents were not near-sighted, nor were other children in the same family. The authors could say little of the rate at which the process developed. One patient who had been observed for eighteen years showed practically no change in refraction from a myopia of 9 diopters. On the other hand, one patient showed an increase of 11 diopters over a period of twenty-one years. It could be said with certainty that the increase in myopia had little to do with the use of the eyes. Practically all exogenous factors could be ruled out.

The authors consider the myopia as strictly axial, since no change in the curvature of the corneal surface and no evidence of changes in the lens curvature or of indicial myopia were found. They regard the characteristic pigmentary changes in the back of the eye as having something to do with the development of the myopia, but in just what way they are not sure. They voice the opinion that the pigment changes are not the result, however, of stretching of the ocular coats. F. H. ADLER.

Retina and Optic Nerve

AN ETIOLOGIC STUDY OF A SERIES OF OPTIC NEUROPATHIES. A. C. WOODS and W. M. ROWLAND, J. A. M. A. **97:** 375 (Aug. 8) 1931.

The authors give the following etiology in a series of 138 patients presenting optic neuropathies, most of whom were admitted to the wards of the Johns Hopkins Hospital on various services. Of these cases, 27.7 per cent were due to actual intracranial tumor, 5.1 per cent to pseudotumor, 17.7 per cent to syphilis, 10.9 per cent to arteriosclerosis, 8.1 per cent to posterior sinus disease, 6.5 per cent to multiple sclerosis, 2.9 per cent to focal infection, 4.4 per cent to toxic amblyopia and 9.1 per

cent to miscellaneous conditions, while in 8.1 per cent the exact etiologic factors could not be determined.

W. ZENTMAYER.

A DEVICE FOR THE ACCURATE LOCALIZATION OF HOLES IN RETINAL DETACHMENTS. R. F. MOORE, Brit. J. Ophth. **15:** 545 (Oct.) 1931.

The device is an adjunct to the means that have been put forward for the accurate localization of the hole or holes in a retinal detachment. It consists of a gold stud, which is inserted through the sclera at the spot that has been calculated, by whatever means, to correspond with the position of the retinal hole. The end of the stud is then identified by means of the ophthalmoscope, and the exact relationship of the retinal hole to it is seen, and the site for the cautery puncture is adjusted in the light of the information so obtained. The studs are made of gold, with a flat circular or oval base and a stem of varying length; a small hole is made in the base through which silk is threaded; otherwise the stud is likely to be lost. The circular base is the better. The base is 5 mm. across. The stem is of four lengths: 2, 4, 6 and 8 mm. At its upper part, it is circular in section and tapers slightly toward its attachment to the base of the stud. If the retinal hole is well away from the choroid, a stud with a long stem is used; if, however, the detachment is a shallow one, a stud with a 2 or a 4 mm. stem is chosen. An incision with a narrow bent needle, with a stop, is now made at the calculated spot, of such a size as will just admit the stem of the stud. Before making the incision, the upper surface of the blade is painted with an alcoholic solution of gentian violet and allowed to become quite dry. The object is to stain the edge of the cut so that the small incision can be easily found again when the needle is withdrawn. The stud is now inserted and pushed right home. The reflected tissues are pulled into place. The relation of the stud to the hole is now studied with the ophthalmoscope. The knowledge that the breadth of the end of the stud is 1 mm. in width assists in the calculation. The stud is removed and the cautery applied at the point calculated for the real location of the tear.

W. ZENTMAYER.

LINDAU, VON HIPPEL AND COATS' DISEASES. A. VASQUEZ BARRIERE, Arch. de oftal. hispano.-am. **31:** 425 (Aug.) 1931.

The author refers to the existing confusion among a certain group of not well classified retinal disturbances, all of a degenerative character with a tendency toward exudation, which affect principally the vascular system of the retina. The typically pure cases present characteristics that permit their being readily distinguished as well defined pathologic entities, but in connection with these characteristics there occur two circumstances that reveal a marked pathogenic relationship: the existence of mixed cases in which lesions of the different diseases coexist and the finding of common anatomopathologic and clinical characteristics which clearly establish their etiologic connection.

Scheerer and Woelfflin divide these cases into two groups: one affecting mostly young persons of the male sex, the other affecting persons of an advanced age, mostly women. The first group, in which the lesions are irregularly distributed in the periphery of the ocular fundus, include

Coats' exudative retinitis, von Hippel's retinal angiomatosis and Leber's retinal degeneration with miliary aneurysms (considered by most authors as identical with the preceding condition). The second group presents most commonly a single central, mostly macular lesion, and includes Fuchs' retinitis circinata and Junius and Kuhnt's disciform degeneration of the central lesion of the retina.

In place of a typical ophthalmoscopic picture characteristic of pure cases, one most commonly finds a disconcerting polymorphism with characteristics belonging to the different varieties. The difficulty in obtaining pathologic material in uncomplicated and comparatively recent cases has not permitted a clear differentiation of the condition from a histologic point of view, but in the few cases examined certain common lesions have been found in the different varieties, such as constant lesions of the retinal vascular system and a tendency toward exudation, which seems to be the primary lesion in some cases and of secondary importance in others.

Lindau's recent studies tend to show that there is no essential difference between the two diseases, which originate in a probably congenital lesion of the vascular system; that this lesion, at least as concerns angiogliomatosis, also affects the vessels of the central nervous system, giving rise to the formation of angiomatous and cystic tumors, which have also been observed in other viscera, such as the pancreas, kidneys, suprarenals and liver—retinal angiomatosis having to be considered not as a local disease but as part of an angiomatosis of the central nervous system. The lesion is very rare, Jess having recently found only about sixty cases reported in the literature, most of them studied only clinically. Coats' disease, although less rare, is far from being frequent.

The author has had the opportunity to study four cases, two of Coats' retinitis and two of intra-ocular pseudotumor, in one of which histologic examination revealed an exudative retinitis with profound angiomatous lesions and in the other a real angiogliomatous tumor of the retina. These cases are studied in detail with drawings and photographs of both the ophthalmoscopic and histologic observations.

The author reviews the writings of Lindau, Cushing, Junius, Rochat and Gourfein-Welt, which tend to show a community of origin and nature in the different varieties of ocular, nervous, cutaneous and vascular manifestations, most probably due to a congenital lesion of the capillary system.

C. E. FINLAY.

GONIN'S OPERATION. M. AMSLER, Klin. Monatsbl. f. Augenh. **86**: 1 (Jan.) 1931.

The author renders a detailed historical account, culminating in Gonin's reasons for devising his operation. De Wecker, in 1870, was the first to point out the frequency of retinal tears and their pathogenic importance in detachment of the retina; Leber, in 1882, drew attention to the action of the vitreous. Gonin attributed the chief action to the vitreous. He found the tear where retina and vitreous had grown together and the detachment resulting through traction from the front. A tear, in Gonin's opinion, is present in every

case of detachment, but it may be located so far forward in the periphery that it cannot be detected with the ophthalmoscope. The tear itself and the examination of the vitreous are of great importance, and therapy must concentrate on attacking the vitreous. The technical questions are given especial consideration in the second chapter of the paper, which contains three subdivisions: (1) examination of the eye, including localization of the tear and selection of the site for the operation; (2) the technic of Gonin's operation; (3) occurrences resulting afterward. The question whether Paquelin's cautery or galvanocautery should be used remains unsettled.

In the third chapter, results and indications are discussed in view of Amsler's personal experiences. He operated on twenty-nine of seventy-four eyes presented, obtaining complete reattachment in twelve. Among the causes for the failures, Amsler considers detachment as a sudden mechanical manifestation of a more or less chronic disease of the eye. Insufficient dilatation of the pupil by mydriatics seems to indicate an unfavorable prognosis, even in the absence of iritis. The prognosis depends on the condition of the vitreous, but not on that of the choroid. Gonin therefore introduces his cautery into the vitreous to unite the changed portion of the vitreous with the scar, thus eliminating all local pulling. It is essential to operate as soon as possible after the detachment occurs.

K. L. STOLL.

A NEW OPERATION FOR DETACHMENT OF THE RETINA. G. GUIST, Klin. Monatsbl. f. Augenh. **86:** 111 (Jan.) 1931.

At a meeting of the Ophthalmological Society of Vienna, held on Dec. 16, 1930, Guist reported a case that gave him the idea for a new operation for detachment of the retina. In an eye with a penetrating wound detachment developed, with a visible tear in the retina. Orbital phlegmon led to hypopyon keratitis, whereupon reattachment followed. The retina and choroid were found grown together, after enucleation that was necessitated by the phlegmon later on. Taking into consideration that necrosis of the retina and hemorrhages with secondary proliferating retinitis were followed by agglutination of the retina and choroid, after Gonin's operation, Guist proceeded to bring about such an inflammation. A rabbit's eye was trephined, and the exposed choroid was touched with a caustic potassium stick for one second. The desired result was obtained—a reactive inflammation without damage to the retina. Cautery with a caustic stick, performed on a vortex vein of another rabbit's eye, produced no hemorrhage, and histologic examination showed the vein to be normal. The caustic stick is embedded in paraffin, which is removed from the tip immediately prior to the operation, and the wound is neutralized with 0.5 per cent solution of acetic acid. Then the choroid is carefully perforated with a conical probe so as to avoid a hemorrhage. The author trephined one rabbit's eye in eight places. He has operated in this manner with satisfactory results in six consecutive cases in human eyes. The tear in the retina of one patient, who was operated on according to Gonin twice before, could be closed. Another patient with myopia of 15 diopters obtained 6/9 vision and could read Jaeger test type 1 five weeks after this method was used for a

detachment following a cataract extraction. In still another patient, in whom a fresh, large tear developed after an operation performed by Gonin personally, no hemorrhage resulted, although a blood vessel crossed the tear.

K. L. STOLL.

Trachoma

IODINE IN THE TREATMENT OF TRACHOMA. J. NEUMANN, Brit. J. Ophth. **15:** 518 (Sept.) 1931.

This paper concerns fifteen cases of trachoma in which the patients were treated with iodine. The conjunctiva is smeared with iodine once a week or once a fortnight; in the interim, a copper sulphate stick or massage is applied to the conjunctiva by the method of Liekernik; i. e., massage with a glass rod with a globular end measuring from 8 to 10 mm. in diameter. No case of artificial injury to the cornea occurred during treatment.

The technic of smearing the conjunctiva with iodine is to wind a wisp of cotton wool around the end of a glass rod and to dip it into a 10 per cent tincture of iodine. The lids are everted, and the iodine is smeared over the tarsal conjunctiva and that of the fornix until the cotton wool is dry. The lower fornix is similarly treated, if necessary. The excess is mopped off with dry cotton wool, and bismuth tribromphenate ointment is applied. The results have always been good.

W. ZENTMAYER.

THE QUESTION OF AUTOSEROTHERAPY IN TRACHOMA. I. A. WASSERMAN, Russk. j. opht. **8:** 89 (Jan.) 1931.

The author treated thirty patients with trachoma by autoserotherapy. Of these, seven were treated by autoserotherapy only. In twenty-three cases combined treatment was used. The eye that was the most severely affected was treated by autoserotherapy, the other eye serving as a control. On the days when subconjunctival injections of the serum were given, no usual treatment was used. Most of the patients suffered from trachoma of the second and third period, complicated by pannus, infiltrations and ulcers of the cornea. The subjective symptoms, the vision and the objective signs improved greatly after the third or the fourth injection whenever combined treatment was used.

The author believes that autoserotherapy is a good auxiliary factor to the usual antitrachomatous treatment. It does not give satisfactory results when used by itself.

O. SITCHEVSKA.

Tumors

A CASE OF DERMO-FIBRO-LIPOMA OF THE CONJUNCTIVA ASSOCIATED WITH OTHER CONGENITAL ABNORMALITIES. H. B. STALLARD and P. P. MARTIN, Brit. J. Ophth. **15:** 580 (Oct.) 1931.

The patient was a boy, aged 3½ years. There was no family history of a congenital anomaly of the parents on either side. In the right eye there was a pinkish-yellow, faintly lobulated swelling extending from the upper to the lower fornix, nearly to the outer canthus and to within

4 mm. of the limbus, except at a position corresponding to 8 o'clock, where a tonguelike process reached the limbic margin. The surface was smooth, with fine hairs projecting from it. The eye was converged. This proved to be mechanical, as it disappeared on removal of the growth. There was marked facial asymmetry, the face being flattened over the right lower jaw, and it seemed on palpation that the junction of the body and the ramus of the mandible on the right side was represented by a gradual curve instead of an angle. There was an accessory auricle situated immediately anterior to the lobule of the right ear. The auricle was well formed, but the external auditory canal was covered over by skin, which extended from the tragus to the concha. A roentgenogram showed the temporary teeth all erupted and normal in number. None of the permanent ones had erupted in either jaw, but the lower first molars were on the point of breaking through. There was a marked asymmetry of the lower jaw, the right side being smaller and less developed than the left. The growth was excised. The histologic diagnosis was dermofibrolipoma of the conjunctiva.

W. ZENTMAYER.

MELANOSARCOMA OF THE CHOROID IN AN ATROPHIC GLOBE WITH OSSIFICATION. G. MARGOTTA, Ann. di ottal. e clin. ocul. **59**: 416, 1931.

The theories explaining bone formation in the choroid, and the cases in which this has accompanied an intra-ocular tumor, are reviewed. The author's patient, a man aged 70, had lost vision in the right eye at the age of 24, at which time an iridectomy was performed. A year before he was seen by the author severe pains developed in this eye. The globe was congested, and smaller than its fellow; the cornea was vascularized, preventing a view of the other media, and the tension was much increased. The presence of a tumor was suspected, and the eye was enucleated. It was adherent to its surroundings, and a fleshy mass of tissue around its posterior pole was removed with it. A recurrence developed in the orbit, and the patient died four months later with symptoms of abdominal metastasis.

Sections showed a large melanosarcoma covering the whole interior of the globe, which had perforated the sclera and surrounded the posterior pole. In the vitreous, anterior to the new growth and connected with it by a thin pedicle of connective tissue, was a semilunar mass of bone in which bone corpuscles and haversian canals were present. In the lacunae was a tissue containing fat cells and enucleated erythrocytes, resembling in every way bone marrow. In some of the bony spaces neoplastic tissue could be seen. No cartilage was found. This bony mass probably originated in the choroid as the result of previous iridocyclitis and was pushed forward into the vitreous by the neoplasm that developed later, leaving only a small pedicle of modified choroidal tissue. Later the neoplasm invaded the bony tissue itself. The relatively early perforation of the sclera, before the globe was filled with neoplastic tissue, was probably due to the previous inflammatory changes, resulting in a lessened resistance of the sclera. It is possible that these old inflammatory changes may have caused the neoplasm to develop.

S. R. GIFFORD.

Uvea

HETEROCHROMIA AND ALLIED CONDITIONS. R. I. LLOYD, Am. J. Ophth. **14:** 287 (April) 1931.

The author considers only the acquired type of heterochromia. He gives a brief history and discusses its relation to the cervical sympathetic nerve, the influence of which was overemphasized. Its incidence in different parts of the world is cited, and attention is called to the fact that although the lighter colored eye is usually affected, in a blond the darker eye may suffer. He discusses the importance of nodules on the pupillary border of the iris as indicating tuberculous disease, which he believes is the usual cause. Four cases are reported and different phases of heterochromia discussed. Atrophy of the iris, melanosis, vitiligo, etc., are then taken up.

W. S. REESE.

Visual Tracts and Fields

THE STRUCTURE OF THE CEREBRAL VISUAL CENTER IN CONGENITAL ANOPHTHALMOS. G. COSMETTATOS, Arch. d'opht. **48:** 282 (April) 1931.

In a previous report the author described the structure of the different ocular residua in a case of congenital anophthalmos. The present paper deals with the histology of the visual cortex in the same case. The numerical study of the cells in the calcarine area of the brain of a patient with anophthalmos and its comparison with those in a normal case showed that except for the first three layers, which contain fewer triangular cells, and the third layer, which has a scarcity of cells, the remaining areas do not differ in structure from the normal. There is also a slight diminution in the size of the first and second layers and especially of the third. In the case reported, the layer of Gennari appeared normal in contradistinction to the reports of other writers. In the external geniculate bodies, the author found the multipolar cells scarce and small, the majority not having reached their full development. The optic tracts and chiasm were reduced to atrophic ribbons free from nerve fibers.

The author discusses his case in relation to those reported by Leonowa, Hanke, Henschen, Bergen, Bolton, Treipel and Lenz. The relationship between the calcarine area of the brain and the retina is also discussed.

S. B. MARLOW.

Therapeutics

OCULAR TUBERCULOSIS. A. N. LEMOINE, Am. J. Ophth. **14:** 439 (May) 1931.

The author believes that tuberculin is often used improperly, and that this accounts for the different opinions as to its value. It is used at too short intervals, thus being given during the negative phase and sensitizing rather than immunizing the patient. The author believes that artificial immunization is a transient process, probably because of disturbed endocrine balance. Before making a diagnostic tuberculin test, syphilis,

focal infection and active pulmonary tuberculosis should be ruled out. It is unsafe to give the test if the patients have a temperature of 99 F. or over. An intracutaneous injection of 0.0001 mg. of old tuberculin is given, and if no reaction ensues in forty-eight hours, 0.01 mg., then 0.1 mg. and finally 1 mg. are given. The temperature should be observed every four hours for twenty-four hours before the tuberculin is given, and for at least seventy-two hours afterward. If there is no increase in the temperature or no focal or local reaction with 1 mg., the lesion is not tuberculous. The first immunizing dose should be about one half of that which gives the first mild reaction. The doses should then be increased about 10 per cent at each injection, at from seven to eleven day intervals, depending on the length of the reaction following the injection. Should the temperature go over 99 F., the dose should be kept the same or diminished. The author discusses various cases in which tuberculin therapy was used.

W. S. REESE.

VITAMIN THERAPY IN OPHTHALMOLOGY. SAL LENCE, Arch. de oftal. hispano.-am. 31: 386 (July) 1931.

The author considers xerophthalmia as due, in a great majority of cases, to avitaminosis.

After extensive considerations of the present status of knowledge of the nature and mode of action of vitamins in normal cases and their therapeutic effect in cases of avitaminosis, and after reference to the best preparations in the market, the author considers that this medication is indicated in lymphatic keratitis, tuberculous keratitis and blepharitis in poorly nourished and debilitated children.

The author reports the case of a girl, aged 14, who had tuberculous antecedents. This patient had double tuberculous keratitis, with almost complete infiltration of the right cornea and loss of vision and less pronounced infiltration of the left cornea without appreciable vision, intense photophobia and marked circumcorneal injection. There were also macrocheilia and polyadenitis of the neck and submaxillary region, simulating adenoma. Under treatment with irradiated ergosterol, the polyadenitis disappeared entirely and the macrocheilia diminished; the right eye was not affected, but the left was improved, with disappearance of the photophobia and lacrimation. After some time the irradiated ergosterol was alternated with polyhormonal treatment. The author finally concludes that vitamins have a marked beneficial action on the ocular disturbances of infancy due to diathetic processes, tuberculosis not excepted; that with their use the duration of the processes is lessened and there is less likelihood of recurrence; that in phlyctenular keratitis their action is specific, and that their value as a reconstituent and regulator of altered metabolism is great.

C. E. FINLAY.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

April 16, 1931

H. MAXWELL LANGDON, M.D., *Chairman*

SPONTANEOUS COMPLETE ABSORPTION OF A CATARACTOUS LENS. DR. T. B. HOLLOWAY and DR. ALFRED COWAN.

J. P., aged 46, a mechanic, married, came to the dispensary of the University Hospital on Oct. 24, 1928, complaining of dimness of vision in the left eye for the past two weeks. He had been unaware of poor vision previous to this time, although he admitted that he had had no ocular examination. There was no knowledge of traumatism. Outside of a gastric ulcer, for which he was operated on in 1922, he had always been in good health. He had been married twenty years, and had had no children. He was the oldest of eighteen children, thirteen of whom died in infancy or early childhood; otherwise, his family history was of no importance.

The pupils were equal and reacted promptly with the various reflexes. In the right eye the vision was 6/6. The media were clear. The disk was healthy and oval, with the upper border slightly blurred. The vessels were somewhat tortuous, and the veins showed some "kinking" where the arteries crossed, indicating early sclerosis. There were no fundus lesions.

In the left eye vision was reduced to light perception. The cornea and aqueous were clear. The undilated pupil was blocked by a cataractous lens. With the pupil dilated, the whole lens appeared shrunken, especially in the anteroposterior diameter, making it wafer-like. When the patient fixed straight ahead, the lens was slightly luxated down and out, leaving a very small, crescentic, dark area in the upper nasal portion of the pupillary space. Physical examination and all laboratory tests revealed nothing abnormal. The patient again denied any knowledge of injury to the eye.

The patient was seen at intervals of about two weeks. The lens slowly but steadily decreased in bulk until, in April, 1929, it was reduced to a thin wafer which sank almost to the lower pupillary border. A good view of the fundus could be obtained. Absorption was now progressing more rapidly, and in May, the small, white disk that represented the remains of the lens was visible only when it bobbed up with marked rotation of the eye. A flat, perfectly transparent, thin membrane across the whole pupillary space, just behind and free from the iris, could be seen with the slit-lamp and corneal microscope. This membrane was not lens capsule, although the surface had a very fine, shagreen appear-

ance and gave a bright iridescence in specular reflection. Numerous tiny white spots were scattered over the surface of this membrane, and behind it, in the vitreous, were two narrow, transparent, scroll-like structures. One of these extended from 8 to 10 o'clock and the other extended from 1 to 4 o'clock in the pupil, 8 mm. in diameter.

In December, 1929, no trace of any part of the crystalline lens could be seen.

On April 7, 1931, the delicate scrolls in the virtreous could still be seen. The thin, transparent membrane, stretching across the pupillary space, lay just behind the plane of the iris. Behind the membrane there was an optically clear space, and behind this space, in the vitreous, were the tubular structures. With the ophthalmoscope these structures appeared transparent. A few fine opacities could be seen in the vitreous. The disk was slightly oval and healthy. There was some pigment roughening around the periphery of the fundus, and in the extreme temporal periphery there was a circumscribed retinochoroidal lesion, measuring about 4 mm. in diameter, with pigment processes, one of which was longer than the others and extended toward the disk along a retinal vessel. The form field showed some nasal contraction. The vision was 6/6 with plus 14, plus 0.25 ax. 45.

The intra-ocular tension had always been normal, but on April 9, it was found to be 36 in the right eye and 33 in the left eye (Schiötz). The patient was given a 0.5 per cent solution of pilocarpine, and on April 13 the tension was 33 in the right eye and 30 in the left eye. At no time were there any other symptoms of glaucoma.

DISCUSSION

DR. T. B. HOLLOWAY: We have watched this case with a great deal of interest at the University Hospital for the past few years, and all of the details are as Dr. Cowan described them. The interesting thing about this case, of course, is the spontaneous absorption. We all know that in many cases of spontaneous absorption one of the interesting phases is the frequency with which it is associated with glaucomatous symptoms. While a certain number of patients may have these symptoms, no active signs were present in our patient.

There was no history of injury to the eye. The patient has been seen at regular intervals, and there has been a slow absorption until at present there is no trace remaining. There is a delicate, practically structureless membrane across the pupillary space, but this membrane is definitely not lens capsule. There are a few fine opacities in the vitreous.

This week the tension, which has been normal, showed evidence of a slight rise, but without any pain or congestive symptoms. At one place in the periphery a delicate pigmentation can be noted overlying a retinal vessel for a short distance.

DR. POSEY: In 1918, I was consulted for glasses by a boy 14 years of age, and at that time nothing abnormal was detected in the right eye other than the fact that vision could not be brought up to more than 5/12 with a correcting lens.

There was no history of traumatism, and when seen the eye was quiet and the lens clear; the pupil the same size as its fellow, and the pupillary border was normal. The iris reacted well to light. The patient was seen again in 1921, and no change in the eye was noted.

He was seen again in 1923, when there were some opacities in the lens, and at that time the boy was turned over to my assistant, Dr. Swindells.

In 1929, I was again asked by the father of the patient to see the boy as he felt that something should be done, when it was found that a cataract had developed on the right eye. There had been no inflammatory symptoms and no rise of tension. In February, 1930, the eye showed a fully developed cataract; the anterior chamber was normal in size, and the iris reacted to light.

The question naturally was as to the cause of the cataract; it was thought possible that there might have been a foreign body going through the lens, but roentgen study showed no foreign body. It was then thought possible that there might be a growth of the ciliary body which pressed on the lens and interfered with its nutrition.

I advised against operation in view of the uncertainty of the origin of the cataract and of what might be back of it. Shortly after this the boy began to notice that he could see out of that eye, and he again consulted Dr. Swindells, who called Dr. John T. Carpenter in consultation, when it was found that the lens was partly subluxated and that there was some vision eccentrically, i. e., counting of fingers when they were held down and out. The eye was quiet. I saw the boy again about a month ago as the vision of the eye had become disturbing because of diplopia; the visual axes were not parallel. The anterior chamber was now found to be deep, the lens shrunken and the pupil practically clear. With a plus 9 diopter lens, the vision came up to 5/15. There had been practically total absorption of the cataract, probably explained by a previous rupture of the posterior capsule of the lens by some blunt force in early life which had left no discernible injury, no evidence of damage to the anterior capsule having been visible at any time.

Dr. William Zentmayer: In November, 1890, I saw in my office a man, aged 62, a typesetter, who came on account of an inflammation in the right eye; he gave a history that the sight of his left eye had begun to fail in 1876. This progressed until at the end of three years he was unable to see more than light. In 1887, he had been told at the Pennsylvania Hospital that he had a cataract and should have it removed.

When I first saw him, the uncorrected vision was 3/150, the sight having gradually improved, and with a plus 11 D. lens it was 5/5, with a 4′ card. In the right eye there was an active iritis, and the lens was cataractous. In the left eye, the media were clear, the disk appeared small, and there was a broad atrophic conus to the temporal side. The fundus was plainly seen with a plus 11 D. lens. The eye was aphakic. When last seen, five years later, vision in this eye was 5/6.

Of course, my case is not as authentic as that of Dr. Holloway and Dr. Cowan, as I did not observe the absorption of the lens, but only saw the end-result. I feel quite certain, however, that traumatism was not a factor.

On another occasion I observed the absorption of a cataractous lens. I saw the child in infancy, when there was a persistence of a hyaloid vessel. The lens at that time was clear. When the child was 5 years of age, the lens became cataractous. When seen after a lapse of several

years, the lens matter had become entirely absorbed, and the hyaloid artery could again be seen extending from the posterior capsule to the disk.

Regarding the development of increased tension in the eye, in my experience this occurs so frequently in aphakic eyes as a late sequel to cataract extraction that I have felt the need of some means of preventing it. This might be accomplished by doing a preliminary iridectomy, making it basal as for glaucoma; it might prove to be a prophylactic measure. I have done this for only about ten years, which is, of course, too brief a time to judge whether it has been of value.

SENILE MACULAR CHANGES OF THE RETINA, ASSOCIATED WITH FOCAL INFECTION. DR. HUNTER SCARLETT.

A review of the literature indicates that macular changes may be associated with focal infections variously distributed throughout the body. Four case histories were presented, each with a diagnosis of senile macular degeneration.

The associated focal infection in the first case was an apical abscess. The second and third patients had diseased tonsils, while the fourth presented a prostatic involvement with a possible dental infection. All these patients had marked retinal arteriosclerosis and vitreous opacities.

The dental roentgen examination in the first case gave negative results, but since the patient had a devitalized tooth on the side of the macular lesion, it was extracted, with a resulting discovery of hemolytic and nonhemolytic streptococci in culture. Vaccines were given, and vision in the right eye, which had dropped from normal to 6/20, returned to 6/10.

One of the patients with an infection of the tonsils, after having been treated with the electric cautery, showed a diminution in vision.

In conclusion, Dr. Scarlett said: Given an eye with markedly diseased retinal vessels, plus a focal infection liberating toxins or bacteria into the blood stream, it is reasonable to expect changes similar to those seen in senile macular degeneration. Vaccines are of aid when focal infections are present.

THE SYPHILITIC OPTIC ATROPHIES. DR. JOSEPH MOORE, Baltimore (by invitation).

A full abstract of this paper appeared in the ARCHIVES (6: 314 [Aug.] 1931).

DISCUSSION

DR. J. H. STOKES: I think that Dr. Moore has made, on the basis of the evidence submitted, a genuine and hopeful contribution to the still very limited control of primary optic atrophy in neurosyphilis. The situation is of interest, particularly because throughout the larger part of the field of neurosyphilology, intraspinal therapy is steadily losing ground and is being replaced by tryparsamide and the various devices for inducing high body temperatures. In fact, though when I first came to Philadelphia I equipped my department at the University Hospital for the administration of intraspinal therapy, I doubt if it has been necessary to treat a dozen patients by this method in the past six years. This, of course, is largely because this form of treatment is not applied

to primary optic atrophy. We shall await with keen interest Dr. Moore's further and confirmatory report on the much larger amount of material of this type that he is handling at the present time. In the meantime, I fear that the individual isolated observer will continue to have the same disconcerting experiences with individual cases that have discouraged us all in the past. Only in larger aggregates and by persistent patient determination can one bring forward such impressive evidence for a relative degree of hopefulness in prognosis in a condition both logically and practically unamenable to treatment. It would seem that services such as that of the Wills Eye Hospital, in which accomplished syphilologists like Dr. Klauder have at their command what must be a very large amount of material, are the proper places, in Philadelphia particularly, for putting this new hope to the test of experience.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Thirty-Sixth Annual Meeting, French Lick, Ind., Sept. 13-19, 1931

MEYER WIENER, M.D., AND BEN WITT KEY, M.D., *Chairmen*

THE SHARE OF THE CRYSTALLINE LENS IN OCULAR REFRACTION. DR. EDWARD JACKSON, Denver.

The records of 644 cases of refraction followed for over ten years were analyzed. In 400 cases, measurements with the ophthalmometer and skiascope allowed an estimation of the part played by the cornea and lens in refractive changes during that period. A slight tendency toward an increase of corneal curvature, and hence of refractive power, was seen in early life. After middle life, such changes were of slight importance. Only 26 eyes showed a change in spherical refraction due to changes in the cornea of over 1 diopter. Forty-four eyes showed changes in corneal astigmatism, but in none was this more than 1.5 diopters. Changes in refraction requiring a change of lens occurred in all but 6 cases. The number of changes in glasses required for any one patient was from 4 to 10, and in nearly all cases this was due to changes in the lens. Slight changes in lenticular refraction are an important cause of eyestrain, and require frequent examinations for their detection.

DISCUSSION

DR. ALFRED COWAN, Philadelphia: The eye of the infant is so short that if it had nearly the refractive power of the adult eye, it would be axially hyperopic 18 or 20 diopters. Since the eyes of infants are generally emmetropic, or nearly so, there must exist, therefore, what might be called a compensating curvature myopia of 18 or 20 diopters. An excess of refractive power of the eye of an infant must exist in the crystalline lens, since the radius of curvature of the cornea of an infant varies little from that of an adult.

It is impossible to ascertain the refracting power of the lens except in the living eye; direct measurements cannot be made with any degree of

accuracy, and the only method left is to calculate it from the loss of refraction that takes place in the eye when the lens is removed. Generally the average aphakic eye requires a correction glass of about 10 or 11 diopters.

Gullstrand, by his exhaustive work, has shown the character of the refraction of the lens. Since the medium of the crystalline lens is not homogeneous, he studied the optical imagery in heterogeneous mediums. He found that, instead of there being a continuously varying index of refraction of the lens substance, the lens consists of a series of isoindicial surfaces of discontinuity, the index of refraction of which increases from the surface to the center, and that during accommodation the lens not only changes its shape, but actually increases its total refractive index.

ASTIGMATISM: ITS ACCURATE DETERMINATION AND CORRECTION. DR. C. S. O'BRIEN, Iowa City.

The author emphasized the importance of astigmatism as the error responsible for nearly all cases of asthenopia. Its accurate correction, he stated, is of far greater practical importance than the correction of spherical errors.

In his belief, skiascopy with cylinders is the most accurate method of determining the axis and amount of astigmatism. The technic of skiascopy with cylinders was described and illustrated. It is better slightly to undercorrect the astigmatism found than to overcorrect it, as the resulting discomfort is less.

DISCUSSION

DR. C. P. CLARK, Indianapolis: The use of an efficient cycloplegic in all patients under the age of 50 years enables me to make a closer estimation of the refractive state of the eye. The examiner should endeavor to determine the near point of accommodation for all patients, for he will occasionally uncover an unexpected degree of ciliary muscle activity in patients who are past 50 years of age. A minimum amount of an effective cycloplegic should be employed in the examination of these patients. A miotic should be instilled into the eye when the examination has been completed to guard against any possible elevation of intra-ocular tension.

The strength and axis of astigmatism can be more accurately determined by skiascopy with combinations of spheres and cylinders than with spheres alone.

DR. LOUIS BOTHMAN, Chicago: Of 1,260 eyes examined under atropine, only 12 per cent required simple spheres; 79 per cent of the eyes in this series showed astigmatism with the rule; this leaves a residue of 9 per cent of the eyes showing an oblique astigmatism, or one against the rule.

The most significant cases in the entire series were those which showed myopia under homatropine, and became mixed astigmatism under atropine. I found this change in 12 per cent of all the myopic eyes in my series. It was also found that 21 per cent of all the cases of mixed astigmatism under homatropine showed hyperopic astigmatism under atropine.

Dr. Edward N. Robertson, Concordia, Kan.: In the case of a cooperative patient with a relatively high amount of astigmatism and clear media, it is not a difficult matter to use the retinoscope and get accurate results; but with mixed astigmatism, especially low degrees, or in a young child, an uncooperative patient or one whose media are not perfectly clear—the very cases in which one must have a reliable objective test—it is not so easy to solve the refractive problem by this method alone. In such cases nothing short of a thorough understanding of the meaning of the varying changes of light and shadows, as well as of the underlying optical principles, will assure success in the accurate estimation of astigmatic errors.

An Experimental Study of Detachment of the Retina and Its Surgical Therapy. Dr. Leo L. Mayer, Chicago.

This article will be published in full in a later issue of the Archives.

Angiomatosis Retinae with Cerebellar Tumor. Dr. Walter S. Atkinson, Watertown, N. Y.

This article will be published in full in a later issue of the Archives.

Visual Fields in Glaucoma: The Effect of Reduced Illumination. Dr. S. B. Marlow, Syracuse, N. Y.

This article appears in full in this issue of the Archives, p. 211.

Malignant Melanotic Tumors of the Eye: A Study of Histologic Types in One Hundred and Eleven Cases. Dr. G. R. Callender, Washington, D. C.

The director of the Division of Ophthalmic Pathology of the American Registry of Pathology at the Army Medical Museum reported on his study of specimens sent to the Museum for diagnosis during nine years. In 1,770 eyes examined, 111 malignant melanotic tumors were found, and subsequent histories were obtained from most of those sending specimens. Four classes of tumors could be distinguished histologically: 1. The Spindle Cell Type. This was divided into subtype A, in which the cells were pointed, showed no definite nucleoli and were arranged in all directions, and subtype B, in which the cells were blunt at one end, which was directed toward a vessel. Less intracellular substance was present, and the cells showed distinct nucleoli. Thirteen cases of subtype A and 22 of subtype B were found. In neither group have any deaths been reported as due to the tumor or its metastases. Less pigment was found in the tumors of subtype B. 2. Epithelioid Type. These showed a predominance of either large or small cells with distinct nucleoli and usually much pigment. Ten cases were reported, with 3 deaths. 3. Fascicular Type. This resembles in structure more an endothelioma. Only 6 tumors of this type were found, of which 2 have resulted fatally. 4. Mixed Cell Type. This showed a predominance of two or more different types in different parts of the tumor. It was the most frequent type seen, and of 60 patients, 15 have died; 11 showed

extension into the orbit at the time of enucleation. Among the 111 cases, 20 deaths have occurred. Dr. Callender concluded that the prognosis for life is better in tumors of the spindle cell type.

DISCUSSION

Dr. Jonas S. Friedenwald, Baltimore: Dr. Friedenwald reminded the Society of the service performed by the Division of Ophthalmic Pathology, which has been given financial and other aid by the Academy. He approved of Dr. Callender's classification, which he compared to Bailey's classification of tumors of the brain according to the stage of development of the dominating cell. He believes that such a basis of classification is more logical than the relative amount of pigment in a tumor, or other criteria. It is logical that tumors of a more primitive cell form should be more malignant than those cells with more nearly mature, but various conditions may create exceptions to this rule.

Dr. V. A. Chapman, Milwaukee: A malignant melanotic tumor of the left caruncle was observed. Recovery was apparently complete five years after removal of the tumor only.

Dr. Martin Cohen, New York: Recently a case was brought to my attention in which there was an iridocyclitis of long standing. The man did not wish his eye removed, and insisted on evisceration of the eyeball. Four months later he developed a growth of orbital tissue which was very hard. General symptoms appeared. Eight months afterward he presented hepatic involvement and also signs in the lungs. I believe that patients with iridocyclitis in whom there is no visibility of the interior of the eye should not be subjected to evisceration because not infrequently in such cases melanosarcoma has been found. A safer procedure is enucleation.

Dr. G. R. Callender, Washington, D. C.: The correlation of the primary tumor in the eye with the metastases is of considerable importance, but unfortunately few autopsies were done in the series of cases reported here.

Cataract in Fishes. Dr. F. Park Lewis, Buffalo.

Investigations were carried out on the form of cataract developing in trout in one of the national museums. The subject had been studied by von Noordman and others, but has attracted practically no attention for the past hundred years. It was found that a species of trematode, or fluke, infests a large proportion of fresh water fish in North America, from 60 to 100 per cent being affected in certain localities. The fluke passes into the water in the excretions of the heron-gull, and goes through an encysted stage in a fresh-water snail, from which free-swimming forms are set free in large numbers. These pierce the skin of fishes and reach all organs, but remain almost exclusively in the eyes. Some are found in the vitreous, but most remain in the crystalline lens which undergoes cataractous change. From forty to fifty flukes are usually found in the lens, but several hundred have been found. Three cases of human cataract have been reported in which flukes were found. The cataractous change is ascribed partly to mechanical irritation by the parasites and partly to a toxic substance secreted by them.

DISCUSSION

DR. HARVEY HOWARD, St. Louis: All the flukes have complicated life cycles. They all require a definitive or primary host, which is generally some vertebrate species: a mammal or a bird. They also require an intermediate or secondary host in the form of a snail, and some flukes require a second intermediate host such as a fish, or another mollusk, or even some form of plant.

There have been recorded a number of cases in human beings with the larvae of certain forms of fluke in the lens of the eye. How did these larvae get there? Drinking the water from ponds or lakes infected with this species of fluke would not apparently bring about an involvement of the lens by these larvae, because the cercariae, or the free-swimming larvae of the kind of fluke (Distoma) that has been found in the eye, cannot withstand the action of the gastric and intestinal secretions.

Recently there have been reported cases of severe dermatitis in man which was caused by the penetration of the skin by the larvae from a certain fluke. Possibly in some of the reported cases of cataract caused by the larvae of the fluke the infection may have originated in that way, i. e., through the skin, while the patient was wading or swimming in a pool infected with these larvae.

THE HYALOID MEMBRANE OF THE VITREOUS. DR. ALFRED COWAN and DR. W. E. FRY, Philadelphia.

Dr. Cowan had previously described a membrane seen after an intracapsular lens extraction in all cases studied with the slit-lamp. This was posterior to the plane of the iris, separating the vitreous from the aqueous chamber. In the present work, the author reports on the dissection of pig, monkey, kangaroo and human eyes.

Two types of preparation were presented. In the first type, after fixation in a diluted solution of formaldehyde U. S. P. (1:10) and hardening in alcohol, the cornea was removed at the limbus, and the iris removed at its base. The anterior portion of the suspensory ligament of the lens was severed and the lens removed in its capsule. Such a preparation examined with the slit-lamp shows a smooth mirror-like surface that has the contour of the posterior lens surface. Behind this surface is a space, optically clear, except for a small number of fiber-like structures, and posterior to this the vitreous structure is seen. Histologic examination of such an eye shows a structure of definite thickness, extending forward from a point just anterior to the ora serrata, coming backward to correspond with the posterior lens surface, and ending at a point on the opposite side of the preparation just anterior to the ora serrata.

In the preparations of the second type the cornea, iris and lens were removed as described. Then the posterior portion of the eye was removed from about 1 mm. behind the ora serrata. Next the vitreous was carefully removed. This left a membrane-like structure which had completely separated the aqueous from the vitreous chamber.

The authors believe that the evidence presented warrants the view that there is a nonnuclear, structureless, uniformly staining membrane

that completely separates the aqueous from the vitreous chamber, that this membrane is normally in contact with the posterior surface of the lens and that it can be designated as the anterior hyaloid membrane of the vitreous.

DISCUSSION

DR. R. C. GAMBLE, Chicago: Embryologic studies indicate that the membrane covering the anterior part of the vitreous is a condensation of the fibrils between the primary and secondary vitreous, and gives no evidence whatever of a hyaloid membrane posteriorly. Histologic examination of human eyes prepared in the usual manner almost always shows a membrane extending across the globe, back of the lens, as described by the authors, and careful observation will show that its anterior surface is smooth, but its posterior surface is somewhat shaggy, due to small fibrils loosely attached to the membrane. The whole membrane is definitely fibrillar; also, the anterior part of the vitreous is especially rich in fibrils, and in this region they tend to be arranged parallel to the plane of the hyaloid membrane. This point is brought out especially in cases in which the vitreous is filled with pus cells. In the anterior part these cells are often arranged in parallel rows between the fibrils, while in other parts of the vitreous there is no special arrangement of the pus cells. My own feeling is that there is an anterior hyaloid membrane that is simply a condensation of the vitreous fibrils.

DR. DANIEL S. KIRBY, New York: There is a condensation layer (hyaloid) representing aggregations of ultramicroscopic micelles limiting the vitreous anteriorly. This layer is in apposition with the posterior lens capsule except for a capillary space which contains a fluid presumably like the aqueous, but which is kept practically obliterated in the intravitreous pressure. The retrolental space, containing primary vitreous, is limited anteriorly by the anterior condensation layer, and posteriorly by the wall of the canal of Cloquet.

DR. ROBERT VON DER HEYDT, Chicago: Dr. Cowan's observations seem to reconcile two controversial conceptions. One of these is that the postlental space is but a capillary slit; the other, that it is quite deep and filled with a thin fluid vitreous, as may be proved by the micro-arc slit-lamp.

I have seen membranes similar to the hyaloid extend upward and join the extraction incision, thus coming anterior to the plane of the iris. It is possible that these membranes are drawn upward by the lens during the extraction, and thus made adherent to the wound. Vitreous is apt to do this. It often follows the knife needle toward the wound and becomes attached to the latter. This is a common slit-lamp observation.

DR. MARK J. SCHOENBERG, New York: The main objection against the current methods of study of the vitreous is the violence we commit by placing the specimen in formaldehyde. If we avoid using hardening fluids, we fail to find the membrane.

I do not say that I deny the existence of the hyaloid membrane, nor do I say there is one. The incontrovertible evidence of a hyaloid membrane, its anatomic demonstration, its specific staining and its separate pathology are still awaiting demonstration.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

H. DICKINSON, *Reporter*

Friday, Oct. 9, 1931

ELMORE BREWERTON, F.R.C.S., *President, in the Chair*

PSEUDOGLIOMA. MR. A. CADDY.

Mr. Caddy saw the boy in May, 1931, at which time he was not complaining of anything, but his mother noticed a white reflex from the left eye. He had had measles two years before. A large mass was to be seen in that eye, growing from the retina and extending into the vitreous. A week before presentation this mass was much as before, and another mass was present on the naso-inferior quadrant of the retina, showing that the inflammatory condition, which the exhibitor considered it to be, was getting worse. Mr. Caddy regarded it as a pseudoglioma, probably due to an obscure infection that may have dated from the attack of measles two years before.

MACROPHTHALMOS. MR. F. W. LAW.

A man, aged 23, complained that the vision in his right eye had always been poor. He had a tremendous globe, and there was myopia to the extent of 24 diopters, with 5 degrees of astigmatism. He could count fingers at 2 feet. There were many vertical ruptures of Descemet's membrane.

COATS' DISEASE. MR. F. W. LAW.

Vision in the right eye was 6/5; that in the left was limited to the ability to count fingers. The tension was normal. In the left eye was a mass of exudate at the macula, presumably a pigment deposit. This mass seemed to be connected to the optic disk by very fine strands, but Mr. Neame regarded it as a purely optical defect. Smaller deposits could be seen round this mass, and many scintillating peripheral deposits two thirds of the way out and upward. It compared well with the third case described in Coats' original paper.

ABERRANT CILIA (?); DERMOID. MR. F. W. LAW.

The boy's mother said that he had always had an extra bunch of eyelashes—apparently he was born with them. Perhaps this was a case of erupted dermoid; the type of hair exactly resembled that on the margin of the lid.

EPISCLERITIS DUE TO FOCAL SEPSIS. MR. A. F. MACCALLAN.

The man had had the disease for thirteen years, during which time he had been submitted to a variety of treatments. He came under the speaker's observation in December, 1930, when he was found to have

a number of septic teeth. These were removed, and antrotomy and turbinectomy were carried out. Since these operations there had been a steady improvement. At the time of presentation the patient had no pain or discomfort, and vision in each eye was 6/6. There had been no optical treatment, beyond a few drops of physiologic solution of sodium chloride.

CHOROIDAL MOLE, BECOMING MALIGNANT. MR. F. E. PRESTON.

The mole was not raised, and there were slight vitreous opacities, which had recently become more pronounced.

DISCUSSION

MR. ELMORE BREWERTON: I counsel watching the case, as it is probably an early melanotic sarcoma.

RECONSTRUCTIVE SURGERY IN THE ORBITAL REGION. MR. T. PROMFRET KILNER.

The first case illustrated the use of the Thiersch graft to provide an eye socket sufficiently large to carry a reasonably sized artificial eye. The first patient, a woman, was injured at the age of 8 years, but Mr. Kilner did not see her for the first time until she was 31. She had had a discharge from the ear, which had been treated by a seton at the back of the neck. The eye she had been wearing was very small, and it was looking almost directly forward. There was practically no sulcus below, and very little above. She had been unable to bring the eyelids forward, but at the time of presentation she could close her lids reasonably over the eye. The patient said that a total of some pints of pus had come out of the back of her neck when attempts were being made to save the sight of the left eye. A large fat graft had been taken from the abdominal wall. Mr. Kilner said that the graft would be further watched. He thought that the final contour of the neck would be satisfactory.

The next case was one of extensive lupus in which the disease had started when the patient was 11 years of age. Treatment was by scraping, and between 1914 and 1924, the patient had been attending the Radium Institute every two months. Mr. Kilner excised the whole of the area indicated; the scar was dragging down the lower eyelid. In one region active ulceration was proceeding; Mr. Kilner did not know whether it was true lupus, or merely the results of the extensive application of radium. He did not expect as good a result from the Thiersch graft as he obtained. The sound principle in these cases, particularly those in which the outlook is doubtful, is to use the most surgically economical method at one's disposal, namely, a Thiersch graft. In the present case, Mr. Kilner did a single Thiersch graft, and it took very well. It not only produced a functional result on the eyelid, but also gave a good cosmetic result on the cheek. There was further ulceration, but not in the graft. Dermatologists opposed the grafting treatment for lupus, as they contended that the disease broke through the graft. In the present case the only breaking through occurred

anterior to the graft. Mr. Kilner put in another graft, and that area also smoothed out and gave a good cosmetic result. There were outlying patches, but these might well have been radium burns.

The next case was also one of lupus, and the patient had ectropion of both lower eyelids, which was worse on the left side. Closure was reasonably good on the right side, but very poor on the left. She had suffered a good deal from epiphora and was very miserable. When Mr. Kilner first saw the patient in 1929, she was an absolute Cinderella, and was undeveloped physically. She still had semiactive lupus and a brawny swelling of the lip with spots of ulceration, and the eyelids were dragged down. Mr. Kilner started with surgical economical replacement by a Thiersch graft, and he showed on the screen the intermediate stages and the result. A mold was used, the eyelid being brought out over the mold, so that the graft was pressed home on the raw surface produced by incising just outside the eyelash margin, allowing the scar tissue to be divided. On the other side, he sewed the eyelids together in their middle two-fourths. The nose was similarly grafted by a Thiersch graft, applied on a mold. Ten days prior to presentation, Mr. Kilner had inserted a small fat graft under the Thiersch graft to fill out the hollow of the eyelid. At the time of presentation, the patient could close her eyes satisfactorily; a photograph enabled the observer to realize the enormous change that had been wrought in the mental attitude of the patient toward life. There had never appeared any suggestion of recurrent lupus in the grafted area.

The next case was presented to show work on the eyelid and the reconstruction of eyebrows. The patient, a man, had been badly burned: he had received from a defective carboy a dose of fuming nitric acid full in the face. Thiersch grafts had been applied on the left eye; a repeat graft had to be used as the first was followed by contraction of the eyebrows; this makes all the difference to faces that have been burned. Mr. Kilner showed pictures of hairy grafts taken from the mastoid region, grafts of full thickness, carrying hair follicles. The grafts were sewn into position and pressure-dressed. The next slide showed a flap that had been brought up in stages from the abdominal wall via the wrist. The flap was brought to the desired position in three stages.

A girl shown in person was one of the few for whom Mr. Kilner had felt justified in trying out a hairy Wolff graft to the margin of the lid. She had a complete line of eyelashes. She had a severe case of epilepsy, and had fallen on a hot iron, badly burning both eyelids. There was a large granulating surface. Mr. Kilner shaved off the granulations, sewed the lids together and placed a Thiersch graft on the raw surface. However, traction occurred, and the lower half of the globe was exposed. Mr. Kilner formed a new lower lid from mucous membrane, and let in a tiny strip of Wolff graft. He tried to reduce the thickness and immobility of the lower eyelid. This case illustrates the disappointing results that one must expect when one has to use a full thickness graft from the scalp to reconstruct an eyelid. In this case particularly, the eyelids seemed to be thick, immobilized masses; however, Mr. Kilner thought that a progressive thinning would

take place. The patient was not making the best of the eyelashes. They grew to a great length, and she cut them occasionally.

One slide showed a case in which the patient was burned by sulphuric acid. There was emmetropia of the upper lid of the right eye, and there were some remains of mucous membrane after a previous treatment, which gave trouble on account of the discharge. A Thiersch graft was applied to the upper lid, and the nose was reconstructed by a Wolff graft, while the angle of the mouth was improved by a small tube pedicle flap brought from the neck. A small tube pedicle was made on the neck, and this was swung up to replace the scar tissue that was causing eversion of the lower lid.

A severe case was one in which the patient had very marked ectropion, with loss of part of the nose and ear. She had epilepsy, and had fallen on a fire; she stayed there until somebody found her. The cheek and nose and been reconstructed, and two pedicle flaps were swung up, in two stages. Thiersch grafts had been applied to the eyelids. She had an excellent eyebrow.

In the next case, the patient was injured in an automobile accident; there was an extensive loss of bone in the orbital and malar region. Most of the malar bone and part of the zygomatic arch had been removed as sequestrums; there was definite diplopia; the eyelid and eyebrow were drooping, and the patient had an ugly scar. Mr. Kilner put in a large fat graft and filled the contour in the malar region. He tried to insert some of the fat graft under the eye. At the time of presentation, the pupils were level, and the eyebrow and eyelid were almost normal.

The next was a very severe and extensive case of lupus, which was shown at Alton by Sir Henry Gauvain. The patient's face was pathetically grotesque. He had had a good deal of treatment with the Finsen light. The nasal openings were stenosed; the eyelids were more puffy than ectropic, and there were extensive corneal opacities. One felt that every effort should be made to secure a covering for the eyes. The patient had some vestige of sight. He was suffering from active lupus. Mr. Kilner let a Thiersch graft into the upper eyelid, doing it on a mold; he did not close the lids together. On the other side, he applied the remains of the eyelids, turning in much conjunctiva and grafting the raw surface. One picture showed the eyelids joined together and a grafted surface covering the lids. The material was present for making a reasonable nose; the upper eyelid was grafted, and the closure was good. The next picture showed a tube pedicle made on the abdominal wall, attached to the wrist, and in that stage being attached to the upper lip. A nose was beginning to form, the eyelids were functioning satisfactorily, and a most hideous looking monster was made much more presentable, as shown by the photographs.

Book Reviews

A Quantitative Study of Achromatic and Chromatic Sensitivity from Center to Periphery of the Visual Field. By Hazel Austin Wentworth. Psychological Monographs. Volume XL. Number 3. Paper. Price, $2.75. Pp. 189. Princeton, N. J.: Psychological Review Company, 1930.

The author worked under Ferree and Rand at Bryn Mawr College from 1922 to 1926. Since then she has held the Fellowship in Ophthalmology in the Medical School of the University of Pennsylvania and the position of Assistant in Perimetry to Dr. Holloway. She now reports her work on the threshold sensitivity of the retina, both achromatic and chromatic. Only one observer was used in these experiments, on which fact the author comments as follows: "While the use of more than one observer in these experiments was precluded by the range of work covered, the validity of result was secured not only through the standardization and control of all factors known to influence the sensitivity of the retina to color, but also through careful tests of the normality of the observer as to acuity, light and color sensitivity and visual fields and of the characteristics of peripheral refraction of the eye employed. In so far, then, as the eye used in investigation may be regarded as representative of the normal eye, and with due regards to individual variation, the conclusions drawn may be considered to have a general application."

The author tested the sensitivity of the retina in a number of meridians and made her determinations as quantitative as possible. Spectrum lights of a definite composition and purity were used as stimuli, and the intensity of these stimuli was expressed in terms of energy. The chromatic thresholds were determined under light and dark adaptations. The intensity of illumination of the former was that which has been adopted as standard in perimetry, namely, 7 foot-candles. The achromatic thresholds were determined with a completely dark adapted eye at the same points in the field as were used for the chromatic threshold. From a comparison of these two sets of threshold values it was possible to designate for the first time in an absolute scale the value of the photochromatic interval. The size of the stimulus used throughout subtended a visual angle of 1 degree and 16 minutes. The energy of the light stimuli was measured at two places, at the analyzing slit of the spectroscope and at the eye by means of a bismuth-silver thermopile and a Thomson galvanometer.

The results obtained by the author are exceedingly interesting and should be studied in detail. A general tendency was found for the area of the color fields to vary with the logarithm of the intensity of the stimulus light. Great irregularities occurred in the ratios of sensitivity from point to point for a given color in the same meridional quadrant and at corresponding points in different quadrants. With stimuli of equal energy for all but very high intensities, an interlacing or crisscrossing of limits

was found for red, blue and green. At all intensities with stimuli equalized in energy, the limits for yellow fell outside those for red, blue and green. The interlacing of limits for red, blue and green is not necessarily a pathologic phenomenon as was formerly and is still believed by some diagnosticians. Contractions of the field, changes in the shape of the field and reversals in the order of ranking as to the breadth of the field also are not necessarily pathologic phenomena. All may be produced for the normal eye by varying any of the factors that strongly affect the retina's response to color. The effective intensity of a 1 degree stimulus of the red and blue of the standard pigment papers (Heidelberg series, the same which are used with the Ferree-Rand perimeter) corresponds closely to an energy value of 100 watts $\times 10^{-13}$ for the spectrum stimuli.

The lowest achromatic sensitivity of the dark-adapted eye to all wavelengths was found at the fovea; the highest, at points ranging from 30 to 50 degrees from the fovea, varying with the color and the quadrant investigated. From these points to the periphery there was a comparatively small decrease in sensitivity. Within the first 5 degrees of the fovea the increase was very rapid (for red 2.18, for yellow 12.52, for green 22.64 and for blue 6.5 times the central sensitivity). The state of adaptation alters the chromatic sensitivity considerably. Within a radius of 30 degrees from the center the chromatic sensitivity of the dark adapted eye is far in excess of that of the light adapted eye. At the center it was from seven to forty times greater, depending on the color, and from 10 to 30 degrees it was from three to fifty times greater. Between 30 and 40 degrees sensitivity to blue and green ends. The sensitivity to yellow and red remained slightly greater for the dark adapted eye up to 50 or 60 degrees.

The author makes no attempt to relate her findings with any of the existing color theories. She points out, however, that irregularity of ratio of sensitivity from point to point in a given meridional quadrant and at corresponding points in the different quadrants and an interlacing of the limits of color sensitivity were found to be typical of the pairs of colors rather than constancy and coincidence of limits, as has been claimed by some of the followers of the paired process theories; also that the irregularity of ratio of sensitivity from point to point in the same meridional quadrant and at corresponding points in the different quadrants makes the possibility of obtaining stable colors, i.e., colors that remain the same in hue from the center to the periphery, to say the least, very remote.

The author concludes that irregularity and incompleteness of development of function are, of course, what might be expected in the peripheral retina from its conditions of use and the service it is called on to render. The actual picture, however, is different from the traditional one drawn to the scale and pattern of theoretical and systematic thinking.

The entire work reported in the monograph bears the characteristics of the school of Ferree and Rand, namely, greatest possible accuracy in all the physical measurements, careful control of the physiologic and psychologic factors involved in the experiments and the objective manner of presentation, free from any speculative tendency.

P. C. KRONFELD.

The Wills Hospital of Philadelphia. The Influence of European and British Ophthalmology upon It, and the Part It Played in the Last Hundred Years in Developing Ophthalmology in America. By Dr. William Campbell Posey and Dr. Samuel Horton Brown. Price, $5. Pp. 340. Philadelphia: J. B. Lippincott Company, 1931.

This book of 340 pages covers much more than its title implies. In the first place, it enlarges and amplifies the history of the famous Wills Hospital that was written by Dr. S. D. Risley in 1908 and published in the hospital report for that year. It presents biographies of past members of the surgical staff with photographs attached. Not only does it trace the development and history of ophthalmology in Philadelphia, but it has much to say in regard to the rise of modern ophthalmology on the continent of Europe and in England. In addition, it touches on our national eye societies, their establishment and growth; it takes up the subject of American journals of ophthalmology, and it deals with the conservation of vision movement in this country, and other kindred subjects.

The history of the Wills Hospital is best read in the biographical notes of its staff. It is the history of ophthalmology in Philadelphia for one hundred years, and to a lesser degree of ophthalmology in the United States. Among the distinguished names of those who have served the Wills Hospital, we may note in passing, are: Isaac Hays, D. Hayes Agnew, George Harlan, William F. Norris, William Thomson, George Strawbridge, Charles A. Oliver, Samuel D. Risley, Lewis Ziegler, Edward Jackson, W. C. Posey and William Zentmayer.

The Wills was one of the pioneer eye hospitals. Of those that endured, only the New York Eye and Ear Infirmary (1820) and the Massachusetts Eye and Ear Infirmary (1824) antedate it. The original hospital was built in 1834 from a bequest of over $100,000 from James Wills, Jr., to the mayor and corporation of Philadelphia to establish a hospital for the blind and lame. It has always been dependent on the city and state for funds, and has always been a purely charitable institution. Beginning with but twenty beds and with no provision for outpatients, there has been a gradual increase from year to year, until in 1930, 2,413 patients were treated in the wards, while the clinic had grown to nearly 25,000 cases.

Throughout the years the Wills Hospital has retained its location on Logan Square, enlarging and modernizing itself from time to time, adding various necessary departments, x-ray, pathologic laboratory, physiologic optics unit, additional operating rooms and pavilions, and has developed a system of associated services to care for the consultation work without which no hospital is properly equipped. Now the time has come when the Logan Square site is no longer adequate for its purposes, and a new building on another desirable location, which will house from 200 to 250 patients, is under construction. In line with modern practice the new building will care for a certain number of private patients. Would that it had been possible at this time to create in Philadelphia an ophthalmological foundation closely connected with one of the strong medical schools similar to those that are now developing in Baltimore, Boston, St. Louis, Iowa City and New York.

In the teaching of ophthalmology, the Wills Hospital has been one of the most important factors in this country. Instruction has been given since its earliest days. It has been a center for postgraduate and undergraduate teaching; many of our best known ophthalmologists have received their training there, and among those who have served on its staff there are many of the leaders of American ophthalmology. The publications of its staff have been many and valuable. That the Wills Hospital has achieved its enviable position in spite of being dependent on the state and city for funds speaks for the public-spirited men who have had its control in their hands, and especially for the quality of its staff, past and present. It has never had the advantages of close connection with a medical school or with a strong general hospital. As an institution it has always stood for a wise conservatism in ophthalmology. As stated before, the history of the Wills Hospital is best read in the biographical sketches of the members of the staff contained in this volume. These can only be read in the original. This book should be widely read, and its authors deserve congratulation and thanks.

G. S. DERBY.

Medical Social Service in Eye Clinics. Transactions of the first of a series of study meetings arranged by the Committee on Development of Social Service in Eye Clinics of the Medical Social Service Section of the Welfare Council of New York City. Pp. 143. New York: Welfare Council of New York City, 1931.

These transactions are made up of a series of lectures given by various New York ophthalmologists to groups of social service workers. Such topics as cataract, glaucoma, refractive errors, strabismus, keratitis and conjunctivitis and the retinal complications of diabetes and nephritis are presented in elementary terms, together with suggestions for the increased usefulness of the social worker in dealing with each of these types of cases. The talks would seem to be admirably adapted to their purpose.

O. P. P.

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Sir J. Herbert Parsons, 54 Queen Anne St., Cavendish Square, London, W. I.
Place: London. Time: July 27-29, 1932.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: May 19-21, 1932.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Rotterdam, Holland.
Place: Madrid, Spain. Time: 1933.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: May 11-13, 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: September 19-23, 1932.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June 27-29, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. David W. Wells, Hotel Westminster, Boston.
Secretary-Treasurer: Dr. Theodore L. Terry, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: Seattle. Time: June 30-July 2, 1932.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternatelv until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. H. L. Harley, 101 S. Indiana Ave., Atlantic City.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.
Place: Atlantic City. Time: June 7-10, 1932.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. R. T. Atkins, 4 W. Fifty-Third St., New York.
Secretary: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Place: Buffalo. Time: May 23-24, 1932.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. D. W. Henderson, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welch, 14 Exchange Pl., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alan C. Woods, 906 Cathedral St., Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. John N. Evans, 23 Shermerhorn St., Brooklyn.
Secretary: Dr. John Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Joseph C. O'Gorman, 1324 Jefferson Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Michael Goldenburg, 104 S. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 Fifth Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. Seventeenth St., Omaha.
Vice President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Julius H. Gross, Carleton Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, 108 W. Sixth St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. Sixth St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.